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NEWER DRUGS IN THE TREATMENT OF HYPERTENSION

IV. USE OF RESERPINE, HYDRALAZINE, AND PENTOLINIUM IN VARIOUS COMBINATIONS

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The possibility of synergistic effects among various drugs used in treating hypertension was explored by testing four combinations: hydralazine and reserpine (18 patients), hydralazine and pentolinium (6), reserpine and pentolinium (30), and all three drugs (24). The dosages varied somewhat, since an effort was made to increase the intake of each drug to reasonable tolerance. The drugs had been used singly in previous studies. The present study indicated the effectiveness of hydralazine when combined with reserpine for treatment of hypertension of moderate severity. Mean blood pressures, as measured in the recumbent position, fell to normal in 11 (61%) of the 18 patients in this group, and the only major drug reaction in this group was hydralazine arthritis in one patient. The combination of hydralazine with reserpine was found to be simple, easily employed in outpatients, and relatively free from intolerable side-effects, provided that the dosage of each drug is gradually adapted to the tolerance of the patient. Hypertensive disease when present in the severe form generally requires the addition of a blocking agent to the combination of reserpine and hydralazine.

DURING THE PAST few years, three new types of drugs have been introduced and utilized in the treatment of primary arterial hypertension, namely, the anticholinergic agents, hydralazine hydrochloride, and various Rauwolfia compounds. When used alone, these drugs have not proved satisfactory as antihypertensive agents for all grades of hypertensive disease because of their inadequate hypotensive action or disagreeable, even intolerable, side-effects when used in effective dosage. The idea of using several drugs in combination,[1] each possessing a different mode of action, in order to produce additive or synergistic effects on blood pressure seems entirely logical. Since the most effective combination of these drugs for the therapy of hypertensive disease is still unsettled, it appeared worthwhile to investigate the effects of reserpine, hydralazine, and pentolinium tartrate employed in various combinations upon a suitable group of hypertensive patients. Pentolinium was the newest of the anticholinergic drugs available at the onset of this study.

Pharmacologically, reserpine, a bradycrotic and tranquilizing agent, produces peripheral vasodilatation by central inhibition of the sympathetic nervous system and possibly by a direct action on the vessel wall.[2] Its principal utility is found in treatment of mild to moderate hypertensive disease. Hydralazine possesses complex actions, including direct peripheral vasodilatation,[3] a weak sympath-

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olytic effect, and neutralization of such pressor principles as hypertensin and pherentasin.[1a] Stimulation of the heart results in increased cardiac output; renal blood flow increases despite fall in blood pressure. When used alone, its hypotensive action is generally weak. Pentolinium,[4] a potent anticholinergic agent which blocks both sympathetic and parasympathetic nervous systems at the ganglion level, achieves blood pressure reduction greater in orthostatic than recumbent positions. Absorption through the intestine is irregular, and wide fluctuations in blood pressure are commonly noted.

Materials and Methods

Previous publications from this hospital have outlined methods of study and follow-up of hypertensive patients undergoing drug therapy.[5] The present series of 78 patients comprises 43 males and 35 females ranging in age from 24 to 65 years (average 45 years). These 78 patients were divided into four groups, each group being treated with a separate combination of drugs. Eighteen patients were treated with reserpine and hydralazine, 6 patients with pentolinium and hydralazine, 30 patients with reserpine and pentolinium, and 24 patients with a combination of all three drugs. Blood pressures, taken in both recumbent and erect positions, were recorded frequently before treatment to establish control levels and repetitively during treatment at daily or weekly intervals.

For purposes of classification in terms of severity of hypertensive vascular disease, patients in this study were divided clinically into four groups—group 1 (mild), group 2 (moderate), group 3 (severe), and group 4 (advanced)—according to a "severity index" similar to those outlined by Page[6] and Goodyer and associates.[7] This severity index is based on level and duration of hypertension, changes in optic fundi and renal function, and the presence of cerebrovascular disease, coronary artery disease, cardiac enlargement, and heart failure. It possesses prognostic significance and offers a simple numerical system to tally changes resulting from treatment. Of the total of 78 patients graded in this manner, 5 were classified as in group 1, 43 in group 2, 17 in group 3, and 13 in group 4.

The three drugs studied had been employed singly or in combination with other drugs in a number of these patients, but inadequate response indicated the need for additional antihypertensive agents. Many patients were transferred from one of the four groups to another because of failure to obtain the desired hypotensive response or because of major reactions to one or more of the drugs. The presence of minor unpleasant side-effects was not an indication for withdrawal of the drug. Wherever possible, blood pressures were recorded at home by a member of the patient's family, since these figures more accurately reflect the average blood pressure than casual blood pressures observed in the physician's office. All recorded blood pressures were included in the averages of reported results. Diets employed varied from the basic rice diet to unrestricted diet. Sodium restriction was ordered for all patients in groups 3 and 4. The majority of patients were treated entirely on an outpatient basis for economic reasons.

Drug dosage schedules for the entire series varied as follows: reserpine, 0.1-0.25 mg. three times daily, increasing to a maximum of 4 mg. daily (average 1.22 mg.); hydralazine, 10-25 mg. four times daily to a maximum of 500 mg. daily (average 259 mg.); and pentolinium, 10-20 mg. four times daily to a maximum of 1,000 mg. daily (average 311 mg.). An attempt was made generally to increase the dose of each drug to reasonable tolerance. In several instances pentolinium initially was administered in parenteral form for short periods before transfer to the oral form. All patients were observed closely for major drug reactions.

TABLE 1.—*Effects of Reserpine, Hydralazine, and Pentolinium in Various Combinations on Mean Blood Pressure**

Fall in Mean Blood Pressure, Mm. Hg.	Reserpine and Hydralazine (18 Patients, 3-12 Mo.)†		Reserpine and Pentolinium (30 Patients, 3-24 Mo.)†		Reserpine, Pentolinium, and Hydralazine (24 Patients, 6-20 Mo.)†	
	Recumbent	Erect	Recumbent	Erect	Recumbent	Erect
0- 9	1	1	6	5	1	6
	11%‡	11%	53%	23%	17%	17%
10-19	1	1	10	2	3	4
20-29	3	4	4	7	9	2
30-39	8 89%	4 89%	6 47%	5 77%	4 83%	5 87%
40+	5	8	4	11	7	13
Patients with fall to normal	11(61%)	13(72%)	1(3%)	13(43%)	7(29%)	14(58%)

*Mean blood pressure is obtained by adding one-third of the difference between the diastolic and systolic pressure to the diastolic pressure.
†Duration of treatment.
‡Percentage figures refer to per cent of patients in drug group.

Results

Reserpine and Hydralazine Therapy.—The group receiving a combination of reserpine and hydralazine comprised 18 patients; initial treatment was begun with reserpine in 13 and with hydralazine in 1. Four were started on therapy with the two drugs simultaneously. There were 10 males and 8 females in this group; their ages averaged 44 years. Duration of treatment varied from 3 to 12 months.

Patients were divided according to severity index as follows: four patients in group 1, nine in group 2, four in group 3, and one in group 4.

Table 1 reveals that 16 patients (89%) exhibited a "definite" fall in mean blood pressure of 20 to 40+ mm. Hg in both recumbent and erect positions. (A fall in mean recumbent blood pressure of 20 mm. Hg or greater is considered a "definite" reduction.) Mean blood pressures fell to "normal" in 11 patients (61%) in the recumbent position and in 13 patients (72%) in the erect position.

("Normal" blood pressure is arbitrarily defined as a mean blood pressure of 115 mm. Hg. or less.) Table 2 presents the fall in blood pressure and mean blood pressure as recorded in recumbent and erect positions and averaged before and after months of therapy for each group. It is noteworthy that the average mean blood pressure for the entire group after reserpine-hydralazine therapy returned to "normal" both in the recumbent and erect positions.

Of the 18 patients treated with these two drugs, the severity index changed as follows: four patients in group 3 reverted to group 2, three in group 2 reverted to group 1, two in group 1 changed to group 2, one in group 2 changed to group 3, and the remaining eight patients were unchanged. The only major drug reaction occurring in this group was hydralazine arthritis in one patient.

Reserpine and Pentolinium Therapy.—The group receiving a combination of reserpine and pentolinium comprised 30 patients; initial treatment was begun with pentolinium in 11, with reserpine in 12, and with both drugs simultaneously in 7. Duration of treatment varied from 3 to 24 months. There were 12 females and 18 males whose average age was 45 years. According to severity index the patients were grouped as follows: 1 in group 1, 16 in group 2, 8 in group 3, and 5 in group 4.

During combined treatment with reserpine and pentolinium, a fall in mean blood pressure of 20 to 40+ mm. Hg was exhibited in 14 patients (47%) in the recumbent position and in 23 patients (77%) in the erect position. Average mean blood pressures fell to normal in 13 patients (43%) in the erect position (table 1) and in 1 patient (3%) in the recumbent position. This indicates the greater postural effects of the blocking agent. The average mean blood pressure fall in the recumbent position was 20 mm. Hg and in the erect position 34 mm. Hg (table 2). However, the resultant mean pressures were still elevated (135 mm. Hg in the recumbent position and 117 mm.Hg in the erect).

The severity indexes changed as follows: one patient in group 4 reverted to group 3, four in group 4 reverted to group 2, three in group 3 reverted to group 2, one in group 2 reverted to group 1, and 21 patients were unchanged.

After varying periods of treatment, 14 of these patients were transferred to the pentolinium-reserpine-hydralazine group because of inadequate hypotensive response and 2 were transferred to the pentolinium-hydralazine group because of reserpine depression. Reserpine therapy was discontinued in one patient because of epigastric distress suggesting peptic ulcer. One patient had all drugs withdrawn because of myocardial infarction, and one patient (group 4) died of cerebrovascular accident after four months of therapy.

Reserpine, Pentolinium, and Hydralazine Therapy.—The group receiving reserpine, pentolinium, and hydralazine comprised 24 patients; initial treatment was begun with reserpine and pentolinium in 14 patients and the three drugs simultaneously in 10 patients. There were 11 males and 13 females whose age averaged 44 years. Treatment time varied from 6 to 20 months. According to severity index, the 24 patients were classified as follows: 14 in group 2, 4 in group 3, and 6 in group 4.

During the administration of these three drugs, mean blood pressure fell 20 to 40+ mm. Hg in both recumbent and erect positions in 20 patients (83%). Mean blood pressure fell to normal in 7 patients (29%) in the recumbent position and in 14 patients (58%) in the erect position (table 1).

After treatment with this combination, reduction in group average mean blood pressure for recumbent and erect positions was comparable to that found in the reserpine-hydralazine group, but greater than that observed in the reserpine-pentolinium group (table 2). It will be noted, however, that the hypertensive disease in the reserpine-hydralazine group was less severe than in the other two groups.

The severity indexes changed as follows: two in group 4 reverted to group 3, five in group 2 reverted to group 1, and one in group 2 changed to group 3. In the remaining 16 patients the severity index remained unchanged.

In this group four patients developed hydralazine arthritis, with L.E. cells in the peripheral blood of two patients, necessitating discontinuance of hydralazine therapy. Reserpine was withdrawn in six patients because of the following major reactions: depression (four patients), anxiety state (one), and distressing nightmares (one). One pa-

TABLE 2.—*Effects of Reserpine, Pentolinium, and Hydralazine in Various Combinations on Group Average Blood Pressure and Mean Blood Pressure*

Drug Combination	Group Av. Blood Pressure, Mm. Hg*				Group Av. Mean Blood Pressure, Mm. Hg					
	Recumbent		Erect		Recumbent			Erect		
	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment	Fall	Before Treatment	After Treatment	Fall
Reserpine and Hydralazine	200/120	156/98	198/119	143/89	147	114	33	144	107	37
Reserpine and Pentolinium	218/125	184/110	207/125	152/100	156	135	20	152	117	34
Reserpine, Pentolinium, and Hydralazine	222/127	176/104	211/127	151/95	158	127	30	155	113	41

* Systolic and diastolic.

tient in this group developed myocardial infarction, and all drugs were withdrawn immediately. Two group 4 patients failed to respond to these three drugs, one dying after 8 months and the other after 13 months of treatment. The deaths of both patients were due to cerebrovascular accidents. The increased incidence of complications in this group were ascribed to the severity of the hypertensive disease and the intensity of treatment with maximal drug dosage for prolonged periods.

Pentolinium and Hydralazine Therapy.—The group receiving pentolinium and hydralazine was comprised of only six patients, four females and two males whose average age was 43 years. Three patients had been initially treated with pentolinium alone, and three were started on therapy with the two drugs simultaneously. As to severity index, they were classified as follows: four in group 2, one in group 3, and one in group 4. Duration of treatment varied from 8 to 14 months.

The average blood pressure for the group fell from 217/116 to 180/106 mm. Hg in the recumbent position and from 203/118 to 166/105 mm. Hg in the erect position. Mean blood pressure fell 20 to 40+ mm. Hg in three of six patients in the recumbent position and in four of six patients in the erect position. The mean blood pressure of one patient fell to normal in the erect position and in one patient in both recumbent and erect positions. This group is too small to permit definite conclusions regarding the effectiveness of this drug combination.

Comment

Hypertensive vascular disease of mild to moderate degree may require no more than small doses of reserpine for adequate control of blood pressure.[9] Constant alertness must be maintained for the development of distressing mental illness, notably, anxiety and depression.[9] With increasing severity of the hypertensive process, satisfactory results are less often achieved with a single drug and further compounds must be added to the therapeutic program.[1] Previous observations on the addition of protoveratrine to reserpine demonstrated no significant hypotensive advantage from the combination.[9] The present study, by contrast, indicates the effectiveness of hydralazine when combined with reserpine for treatment of hypertension of moderate severity. The combination is simple, easily employed on an outpatient basis, and relatively free of intolerable side-effects if gradual titration of both drugs is accomplished. Caution must be observed for the development of bone marrow depression or the various clinical features of the "collagen-like" syndrome (fever, chills, arthritis, pleuritis, pericarditis and L.E. cells in blood), which generally occur only when dosage exceeds 400 mg. per day for periods of over six months.[10]

When pentolinium was used in combination with reserpine[14] the effects appeared less satisfactory than those achieved by the reserpine-hydralazine combination. This difference is due to the greater number of severely hypertensive patients in the reserpine-pentolinium group. The addition of the blocking agent generally added minor side-effects, such as blurred vision, dry mouth, and constipation. Irregular absorption from the intestine led to wide fluctuations in blood pressure, both recumbent and orthostatic. Because of inadequate response, hydralazine was added to the reserpine-pentolinium treatment program of 14 patients. Greater fall in mean blood pressure resulted and more satisfactory control of blood pressure followed, of degree closely approximating the results obtained in 10 patients to whom all three drugs were administered initially. Results obtained by the combination of hydralazine and pentolinium were less satisfactory, but the group was too small for adequate evaluation. It would seem worthwhile to use reserpine and hydralazine in combination when more than reserpine alone is needed. When preliminary observations indicate that more potent compounds will be needed for patients exhibiting the more severe grades of hypertension (i. e., groups 3 and 4), therapy may be initiated under hospital control with reserpine and a blocking agent, hydralazine being added as soon as blood pressure fluctuation from the blocking agent is obtained. An alternative method on an outpatient basis consists of initial titration of reserpine and hydralazine dosage, adding the blocking agent if satisfactory results are not obtained with two drugs alone. The anticholinergic drugs are the most difficult to regulate properly and offer the greatest potential hazards for patients suffering from cerebral and coronary arteriosclerosis and renal insufficiency. Gradual titration of the dosage of each drug cannot be overemphasized in the achievement of successful results. Certainly additive effects are obtained when two or more drugs having different sites of action are used together. Amelioration of side-effects is noted from reserpine in combination with other drugs. After hypertension has been controlled for several months, it is often possible to lower the dosage of each drug and, in a few patients, withdraw one drug entirely.

Summary

Seventy-eight patients suffering from hypertensive vascular disease of varying severity were treated with different combinations of reserpine, hydralazine, and pentolinium. Eighteen patients exhibiting hypertension of moderate severity received reserpine and hydralazine in combination. A definite reduction in mean recumbent blood pressure was observed in 16 patients (89%) and

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normal blood pressure was achieved in 11 patients (61%). This combination of drugs proved effective for this group of patients.

Thirty patients exhibiting hypertensive disease of greater severity were treated with reserpine and pentolinium. The mean recumbent blood pressure fell definitely in 14 patients (47%), and in one it reached the normal range. Lack of satisfactory blood pressure control necessitated the addition of a third drug (hydralazine) to the therapeutic program of 14 patients.

Twenty-four patients who had moderate to severe hypertensive disease were treated with three drugs, reserpine, hydralazine, and pentolinium, in combination. Definite reduction in mean recumbent blood pressure was noted in 20 patients (83%), and in 7 patients (29%) the mean blood pressure reached normal values. Comparable results were obtained in one group (14 patients) to which a third drug was added and another group (10 patients) in which therapy with all three drugs was started simultaneously. In a small group of six patients treated with hydralazine and pentolinium, less satisfactory results were achieved, but the number of patients is too small for decisive conclusions.

For hypertensive disease of moderate degree not satisfactorily controlled by reserpine alone the addition of hydralazine has proved to be a simple and effective measure. Hypertensive disease, when present in severe form, generally requires the action of a blocking agent in combination with reserpine and hydralazine.

The reserpine and hydralazine used in this study were supplied as Serpasil and Apresoline by Ciba Pharmaceutical Products, Inc., Summit, N. J.; the pentolinium was supplied as Ansolysen by Wyeth, Inc., Philadelphia.

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DUMPING SYNDROME.—The dumping syndrome is a symptom complex appearing in some patients after gastric resection; it is characterized by epigastric discomfort, nausea, palpitation, tachycardia, weakness, syncope, often a feeling of warmth, and sometimes explosive diarrhea. Rapid gastric emptying results in intrajejunal hyperosmolarity, with a compensating shift of extracellular fluid and plasma into the jejunal lumen to achieve isotonicity. Consequently, a reduction in circulating plasma volume occurs. . . . Increased peripheral blood flow is present in many patients with dumping symptoms. . . . The increased peripheral blood flow is closely related to the systemic symptoms of the dumping syndrome, such as syncope, tachycardia, and weakness. It is suggested that patients who exhibit severe systemic manifestations in dumping attacks have abnormal homeostatic vascular responses to sudden reductions in plasma volume.—D. B. Hinshaw, M.D., and others, Peripheral Blood Flow and Blood Volume Studies in the Dumping Syndrome, *A. M. A. Archives of Surgery*, May, 1957.

SERUM CHOLESTEROL MEASUREMENT—HAZARDS IN CLINICAL INTERPRETATION

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The conclusions that can be drawn from determinations of total serum cholesterol must take into account the varying results obtained by the use of different methods in different laboratories, the discrepancies to be expected between samples taken on the same day, and the physiological variations in a given individual over periods of weeks or months. Duplicate serums from three patients who had had myocardial infarction were analyzed in five different laboratories, one of which used two methods. Not only were the results consistently higher in some laboratories than in others, but the rank-order of the results varied also. The discrepancies were such as to necessitate great caution in drawing inferences regarding diagnosis and treatment from a single cholesterol determination. In assessing the patient's progress on therapy, to be of significance a cholesterol level checked but once after therapy must decrease about 100 mg. per 100 cc. or else there must be a lesser decrease of about 60 mg. per 100 cc. which is consistently maintained.

Much has been written concerning the relationships between serum lipids and lipoproteins and atherosclerosis.[1] It is not the purpose of this report to discuss the evidence for or against these relationships or to compare the relative merits of the various kinds of lipid measurements. Rather, we propose to examine the reliability and the clinical interpretation of one of these measurements, that of total serum cholesterol.

A recent exhaustive report states in its summary, "The measurement of total serum cholesterol remains the most practicable laboratory measurement for aiding in the identification of people with gross disturbances of lipid metabolism which predispose to coronary disease."[2] Keys' experience essentially agrees with this opinion.[3] He has urged that a serum cholesterol measurement be made a part of the annual physical check up, particularly in men over 30, in order to select the individual who should observe special diet precautions or receive special therapy.

If the physician in practice is to heed this advice he should know the answers to the following questions: 1. What is the reproducibility of the results of a given cholesterol method in a specific laboratory? With what confidence can the clinician assume that a single cholesterol measurement represents the true level? 2. What is the range of variability of duplicate samples performed in different laboratories and by different methods? 3. What are the ranges of biological fluctuation of cholesterol levels over a period of months in patients with proved atherosclerosis who do not receive treatment? This paper attempts to answer these questions.

Materials and Methods

The 10 patients who form the basis for this report are part of a larger study inquiring into the long-term effects of various agents on the clinical course of disease in persons who have survived an acute myocardial infarction. This group ranged in age from 40 to 60 years. No patients with complicating diseases, such as diabetes, hypertension, thyroid or renal disease, or xanthomatous abnormalities, were included. These 10 candidates for placebo treatment were randomly selected by choosing hospital registration numbers ending in odd digits. Careful inquiry into diet disclosed no substantial change during the observation period from that before infarction occurred. All of the men were eating the usual American diet, estimated to contain 35-45% of the calories from fat. No patient showed a change in weight of more than 5 lb. (2.3 kg.) during the study. No other treatment was given. Total serum cholesterol measurements were made on patients after a 12-hour fast, the first being done at least two months after the infarction and thereafter at monthly or more frequent intervals for 6 to 12 months. Measurements were made by the Kingsley method,[4] in duplicate, on the same day. Other replicate measurements were made one week to one month later on serum stored in the refrigerator at temperatures of 0 to 6 C.

In order to learn what variability in results might be encountered by analysis of duplicate serum samples in various laboratories, the following program was devised. Three sets of "blind" duplicate samples were submitted to five laboratories on different days. These laboratories were chosen as represen-

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tative of the facilities available in this community and included a Veterans Administration hospital clinical laboratory, a university hospital clinical laboratory, two reputable commercial laboratories, and our own research unit.

Results

Our own laboratory has used the Kingsley method to perform a total of 1,500 replicate measurements on 500 serum samples. When a given serum sample is measured on the same day and in the same "run," analysis shows a respectable average standard deviation for duplicates of ± 16 mg. per 100 cc. The record is less reassuring when the repeat measurement of the sample is performed a day, a week, or a month later. We have no evidence that cholesterol is "lost" or destroyed during storage in the refrigerator. However, when the reproducibility is reanalyzed under these circumstances, the average deviation is found to be ± 27 mg. per 100 cc.

Results obtained in the interlaboratory study are listed in table 1. The most striking thing to be noted is the extreme range of the results. Sample A-B is reported to contain from 311 to 598 mg. per 100 cc. of cholesterol, sample C-D from 183 to 296 mg. per 100 cc., and sample E-F from 172 to 312 mg. per 100 cc.

By simple inspection it is apparent that results reported from commercial laboratory no. 2 are consistently and substantially lower than those reported from the other laboratories. Excluding this laboratory, the standard deviation for all other laboratories is similar and averages ± 28 mg. per 100 cc. for a single determination. This figure is nearly identical with the ± 27 mg. per 100 cc. obtained from the larger study in our laboratory.

TABLE 1.—*Interlaboratory Comparison of Cholesterol Levels (Milligrams per 100 Cc.) and Reproducibility*

Serum	VA Hospital*	Authors' Laboratory*	University Hospital†	Commercial Laboratory‡		Commercial Laboratory§
				Method 1†	Method 2‡	
A	479	529	480	598	513	311
B	418	487	541	500	453	319
C	240	300	255	291	273	183
D	233	273	256	263	272	191
E	213	218	275	312	255	180
F	220	249	288	252	246	172

* Method of Kingsley and Schaffert.[4]
† Method of Bloor: Modified Methods, in Fister, H. J.: Manual of Standardized Procedures for Spectrophotometric Chemistry, New York, Standard Scientific Supply Corporation, 1950.
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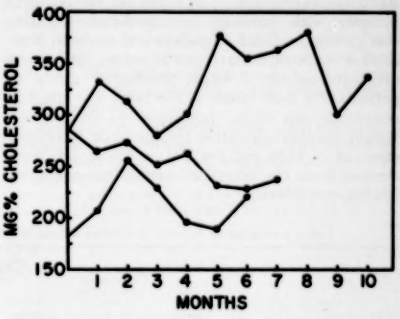
Depicted in the figure are the month-to-month levels of serum cholesterol in representative patients treated with placebo in the high, middle, and low concentration ranges. Table 2 indicates the data for all 10 patients receiving placebo medication. The reader's attention is called to the wide fluctuation of the serum cholesterol level in some patients and the negligible amount in others and to the weighted average standard deviation of ± 30 mg. per 100 cc. biological variation. Although there is not a wide spread in mean cholesterol concentration in this group of patients, analysis failed to show any significantly greater variation at the higher over the lower levels.

The magnitude of change of serum cholesterol level which would be of statistical significance at the 95% level of confidence (less than 1-20 odds of the observed difference being due to chance) was



Serial serum cholesterol levels in three representative patients treated with placebo.

computed. Under the conditions of this study, this figure is ± 112 mg. per 100 cc. for a single determination and ± 100 mg. per 100 cc. when the measurements are made in duplicate. These figures are based on the sum of the variances (squares of the standard deviations) when the laboratory variance is added to the individual biological variance adjusted for laboratory error.

Comment

Had a physician in the course of an annual physical examination submitted sample E (table 1) to commercial laboratory no. 1, he might have felt compelled by the reported serum cholesterol level of 312 mg. per 100 cc. to recommend special dietary restriction for his patient. On the other hand, had the same sample been sent to commercial laboratory no. 2 (180 mg. per 100 cc.), he might have reassured his patient that his serum cholesterol level was well down in the normal range and that he was therefore an unlikely candidate for coronary disease.

Likewise, had a sample been submitted to commercial laboratory no. 1 on the day the E "run" was made (312 mg. per 100 cc.), the physician's course of action may have been quite different from that he would choose had the same sample been submitted to the same laboratory on the F "run" day (252 mg. per 100 cc.). The VA physician submitting sample A to his hospital laboratory and receiving a report of 479 mg. per 100 cc. might be impressed with the results of sitosterols or other agents when

the serum cholesterol level two weeks later proved to be 418 mg. per 100 cc., if he were unaware that this difference is within the range of his laboratory's technical error.

Although the serial fluctuations of serum cholesterol level have been well studied in normal persons,[3] periodic changes in the levels of patients with atherosclerosis have not had sufficient attention. Steiner and Domanski [4] have reported a mean standard deviation of ± 24.8 mg. per 100 cc. in 15 patients with coronary arteriosclerosis; Watkin and co-workers [5] did a fundamental study to demonstrate biological variations in serum lipid measurements among a large, unselected group of patients. For individuals in the same age range as covered by our study, they report an average biological standard deviation (exclusive of laboratory error) of ± 13.96 mg. per 100 cc. These data were derived from cholesterol concentrations averaging 208 mg. per 100 cc.

Our own data indicate biological fluctuation to be somewhat higher. Perhaps this difference exists because all of our patients had had myocardial infarction and their mean serum cholesterol level was 77 mg. per 100 cc. greater.

While this evidence indicates that a shift of 100 mg. per 100 cc. in total serum cholesterol concentration is necessary before one can ascribe a result to treatment rather than chance, such a magnitude of change is not necessary for all persons. Table 2 indicates that some persons have remarkably little biological fluctuation. Unfortunately, the clinician cannot know in which patients the determinations will be relatively stable without first measuring control cholesterol levels for several months. Additionally, since the month-to-month measurements fluctuate without apparent trend, it is possible that a drop in serum cholesterol level of less than 100 mg. per 100 cc. which remained at the lower level on repeated determinations would represent a significant result. If one strikes an average of the standard deviations in patients showing the least biological variation and adds to this the laboratory error, then the 95% confidence limits for statistical significance in this situation would be a change of ± 60 mg. per 100 cc.

It should be emphasized again that the ± 27 mg. per 100 cc. standard deviation figure for laboratory error or reproducibility applies only to the laboratories and techniques involved in this study. Special laboratories might show a better record, as the report of the technical group of the committee on lipoproteins and atherosclerosis indicates.[6] However, our data will serve as a guide for the clinician using the ordinary commercial or hospital laboratory.

Summary and Conclusions

There is a wide discrepancy in results of serum cholesterol measurements performed in different laboratories. The reproducibility record of a given laboratory and a given method should be evaluated on the basis of replicate measurements made on different days rather than during the same "run." The identity of duplicate samples should not be available to the technician.

The biological fluctuation in total cholesterol levels of the patients with atherosclerosis treated with placebo in this study averaged ± 30 mg. per 100 cc. standard deviation.

The physician who orders a serum cholesterol analysis to assess "atherogenic potential" should (1) know the performance of the laboratory he selects, and (2) ask the laboratory to submit its standard deviations of reproducibility (performed as described above) along with the report. The physician who prescribes dietary or other treatment for patients with elevated serum cholesterol levels and who wishes to follow results intermittently should use the same laboratory throughout. He should recognize that, to be of significance, a cholesterol level checked one time after therapy must decrease about 100 mg. per 100 cc., or else there must be a lesser decrease of about 60 mg. per 100 cc. which is consistently maintained.

TABLE 2.—*Range of Serum Cholesterol Levels and Statistical Derivations Of Ten Placebo-Treated Patients**

Case No.	Age, Yr.	Length of Observation, Mo.	Cholesterol Level, Mg./100 Cc.		Maximum Deviation from Mean, %	Biological Variance		
			Range	Mean		Total Variance	Total Minus Laboratory Variance	Standard Deviation
1	56	6	253-345	292	18	1,587	1,260	36
2	40	12	278-373	328	15	1,620	1,388	37
3	52	6	303-392	361	16	1,381	1,039	32
4	41	6	229-299	268	14	1,830	1,678	41
5	59	8	236-286	278	22	462	169	13
6	60	7	182-252	214	18	1,174	847	29
7	43	6	172-286	243	29	321	230	5
8	55	6	280-355	311	14	566	455	21
9	59	7	240-309	292	27	1,860	1,734	42
10	41	6	241-287	266	9	83	0	0
Av.				285	18		845	
Weighted av.							929	30

* No trend could be noted; rise and fall appeared at random.

The flood of literature recommending various agents to lower serum cholesterol should be appraised with these data in mind.

This study was supported by grants from the G. D. Searle Company, Chicago, and the County Heart Association, Riverside, Calif.

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ANTIHYPERTENSIVE DRUGS AS A CAUSE OF THE ACUTE ABDOMEN

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and
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Antihypertensive drugs may produce acute abdominal problems. Since one of us (W. F.) within a period of a few months has been asked to see, for evaluation of a possible acute surgical abdomen, four patients receiving antihypertensive drugs, it seems worthwhile to reemphasize the possibility of abdominal complaints occurring during such treatment.

Four patients, with a clinical picture suggesting the possibility of an acute surgical abdomen to the extent of prompting surgical consultation, were eventually diagnosed as having paralytic ileus and/or vomiting from antihypertensive drugs. Diagnosis of such side-effects depends on an awareness that the patients are receiving the drugs.

Report of Cases

CASE 1.—On June 27, 1956, a 49-year-old man was admitted to the hospital.

History.—The patient stated that for three to five weeks prior to admission he had become progressively short of breath and unable to continue his duties as a mail carrier. For a period of seven months he had had increasing pedal edema. He had been hospitalized in 1952 for similar complaints. Before the present admission he had been taking digitalis and had lost 20 lb. (9.1 kg.) by following a cardiac diet.

Physical Examination.—This well-developed, well-nourished, middle-aged, bed-ridden man appeared to be very apprehensive and dyspneic. Blood pressure was 260/160 mm. Hg. Moist rales were heard posteriorly at both lung bases. The heart was enlarged to the left. The liver was palpable 2 fingerbreadths below the right costal border. A 2+ pitting edema was present over both lower extremities.

Laboratory Data.—The complete blood cell count was essentially normal. The urinalysis was normal except for an albumin level of 30 mg. per 100 cc. and, on microscopic examination, a rare erythrocyte.

Hospital Course.—Initially, the patient's cardiac decompensation was treated with a diet limited to 1,500 mg. of sodium, 4 cc. of elixir of potassium gluconate (Kaon) three times a day, and 0.5 mg. of amorphous gitalin (Citaligin) daily. His severe hypertension persisted.

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On July 6, administration of 2.5 mg. of mecamylamine (Inversine) hydrochloride and 25 mg. of hydralazine hydrochloride four times daily was started. The dosages of these drugs were slowly increased, and, on July 13, administration of 0.25 mg. of reserpine (Serpasil) four times daily was begun. By July 23, the dosage of mecamylamine hydrochloride had been increased to 15 mg. and the hydralazine hydrochloride to 100 mg. four times daily. The evening of that day the patient began to complain of constipation and to become moderately lethargic.

On July 27, the patient was very restless, his abdomen was becoming distended, and his blood pressure had gradually fallen to 150/100 mm. Hg. All drugs except gitalin were withdrawn, and administration of 5 mg. of pilocarpine nitrate three times daily was started.

On July 28, abdominal roentgenograms (fig. 1, *left*) were read as follows: "Erect and recumbent films of the abdomen show a rather diffuse accumulation of gas in the small gut over virtually the entire upper abdomen and to a lesser extent in the lower abdomen consistent with small bowel obstruction. No gas is noted in the large bowel." A Miller-Abbott tube was inserted, and on the following day another roentgenogram (fig. 1, *right*) was interpreted as follows: "A Miller-Abbott tube is coiled in the stomach with the degree of distention of the loops increased over the previous film."

By Aug. 1, the abdomen was less distended but active intestinal sounds could still not be heard. From then on, the ileus slowly disappeared, and the patient was discharged on Sept. 1, 1956.

Final Diagnoses.—Final diagnoses were (1) paralytic ileus resulting from use of antihypertensive drugs and (2) hypertensive cardiovascular disease.

Case 2.—A 67-year-old man was admitted to the hospital on Sept. 23, 1956.

History.—This man, who had been confined to the hospital for three months, had been treated for a variety of conditions. At the time of admission, he had a left-sided paralysis due to a cerebrovascular accident on the right. For years he had had hypertensive vascular disease with a blood pressure of about 210/130 mm. Hg.

On Dec. 26, 1956, the patient had a sudden rise in blood pressure to 260/160 mm. Hg. During the next 36 hours he received 10 mg. of hexamethonium chloride in divided doses. The blood pressure dropped to 140/90 mm. Hg. Then he was given 2.5 mg. of mecamylamine hydrochloride three times daily and 5 mg. of bethanechol (Urecholine) chloride twice daily. With such medication, the blood pressure stabilized at 150/100 mm. Hg, but, on the evening of Dec. 30, the patient began to have distention of his abdomen and to vomit.

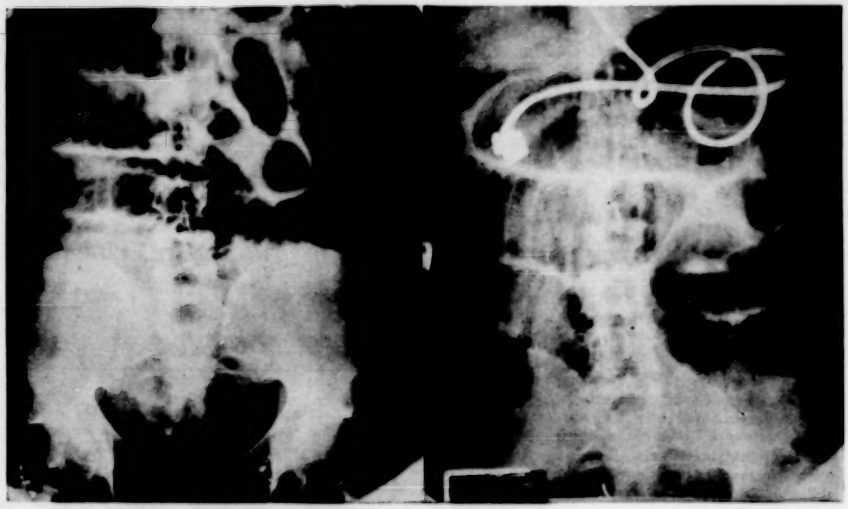

Fig. 1.—Abdominal roentgenograms of patient in case 1. *Left,* roentgenogram made when surgical consultation was first requested. Note the dilated loops of small intestine. *Right,* roentgenogram made one day later. The small intestinal loops are more dilated.

Physical Examination.—On the evening of Dec. 31, the patient appeared to be acutely and chronically ill and somewhat disoriented. His temperature was 98.4 F (36.9 C), pulse rate 104, and blood pressure 150/110 mm. Hg. There was slight paralysis of the muscles of the left side of the face. The abdomen was moderately distended, and no peristaltic sounds could be heard.

Laboratory Data.—The total white blood cell count was 14,000, with 20% nonsegmented neutrophils, 64% segmented neutrophils, 3% eosinophils, 11% lymphocytes and 2% monocytes. On the evening of Dec. 31, abdominal roentgenograms (fig. 2) showed dilated small intestine with fluid levels and considerable gas and feces in the large intestine.

Hospital Course.—A Levin tube was placed in the stomach; intravenous administration of fluids was started slowly; and all medication was discontinued. Within less than a week after the onset of the abdominal symptoms, the patient was again able to eat normally and was having spontaneous bowel movements.

Final Diagnoses.—The final diagnoses were (1) paralytic ileus resulting from the use of hexamethonium chloride and mecamylamine hydrochloride and (2) hypertensive vascular disease.

Case 3.—A 64-year-old man stated that at about 12:30 a. m., Nov. 21, 1956, he developed some vague abdominal pains which soon became sharp and generalized. During the day he vomited several times. Two enemas failed to result in the passage of any gas or feces.

History.—When the patient had been under care for acute pulmonary edema in Dec., 1955, he had had a blood pressure of 240/140 mm. Hg. During the first part of 1956 he had been treated with reserpine, hexamethonium chloride, and hydralazine hydrochloride. From May, 1956, to the time of hospital admission, he had been taking mecamylamine hydrochloride. At the time of hospitalization, he was taking 10 mg. of the latter drug three times daily and, sporadically, 5 mg. of pilocarpine nitrate twice daily.

Physical Examination.—The patient appeared to be having a moderate amount of generalized, crampy abdominal pain. His temperature was 98 F (36.7 C), pulse rate 98, and respiratory rate 22. The blood pressure was 230/110 mm. Hg. The abdomen was moderately distended with some hyperactive intestinal sounds.

Laboratory Data.—The hemoglobin level was 13.3 Gm. per 100 cc. The total leukocyte count was 10,350 per cubic millimeter with 41% nonsegmented neutrophils, 28% segmented neutrophils, 4% eosinophils, 19% lymphocytes, and 8% monocytes. Urinalysis was not remarkable. Roentgenograms were interpreted as follows: "Films of the abdomen demonstrate a moderate amount of air in both large and small bowel. A large amount of fecal material is present in the colon. The films from our point of view are not sufficient for the diagnosis of a mechanical bowel obstruction."

With discontinuation of all medication and enemas, the patient's condition rapidly improved, and he began to have spontaneous bowel movements. He was discharged on Nov. 24, 1956.

Final Diagnoses.—The final diagnoses were (1) vomiting resulting from use of mecamylamine hydrochloride and (2) hypertensive cardiovascular disease.

Case 4.—A 46-year-old woman was admitted to the hospital on Feb. 7, 1957.

History.—The patient stated that about six hours prior to hospital admission she developed crampy upper abdominal pain and began to vomit. She had been constipated for several days.

For some years she had had hypertensive vascular disease with her blood pressure about 200/130 mm. Hg. In early 1956, she had been given hexamethonium chloride, and in the latter part of 1956 and early 1957, mecamylamine hydrochloride. During the month prior to hospitalization, she had taken 15 mg. of mecamylamine hydrochloride daily with two 5 mg. and two 2.5 mg. doses per day.

Physical Examination.—The patient was fairly well developed, slightly obese, and was having abdominal pain. Her temperature was 99.4 F (37.4 C), pulse rate 76, respiratory rate 20, and blood pressure 140/90 mm. Hg. There was slight generalized abdominal tenderness, and peristaltic sounds were hypoactive.

Laboratory Data.—At the time of the patient's admission the complete blood cell count was essentially normal. The urinalysis was negative except for 1+ albumin and granular and hyaline casts. Abdominal roentgenograms were interpreted as follows: "The stomach is distended with fluid and gas; there is tendency to ileus as evidenced by numerous small segments of bowel partly filled with gas and fluid."

Hospital Course.—The patient was given glucose intravenously for nourishment, and the mecamylamine hydrochloride was withdrawn. On Feb. 8, the day after admission, the blood pressure was still 140/90 mm. Hg, but on Feb. 9, the blood pressure had risen to 190/100 mm. Hg. By Feb. 8, the patient was free of pain and was hungry; she was placed on a low-salt diet; she continued to improve and was discharged on Feb. 9, 1957.

Final Diagnoses.—The final diagnoses were (1) vomiting resulting from use of mecamylamine hydrochloride and (2) hypertensive vascular disease.

Fig. 2.—Abdominal roentgenogram of patient in case 2. Note generalized distribution of dilated loops of small intestine and gas in sigmoid colon.

Comment

In these four cases the clinical picture suggested the possibility of an acute surgical abdomen and prompted surgical consultation. In case 1, the patient's abdomen became quite distended, and he did not have bowel movements; in addition, abdominal roentgenograms indicated numerous loops of dilated small intestine (fig. 1). In case 2, the distention was not quite as severe as in case 1, and

the roentgenograms (fig. 2) showed considerable gas and feces in the colon. In cases 3 and 4, the patients were not so sick; they complained primarily of abdominal pain and vomiting.

Diagnosis of such side-effects with the antihypertensive drugs depends considerably on an awareness that patients receiving such drugs may develop complications as those described in the four cases. If it is not realized that such complications may occur with these drugs, early and accurate evaluation of such abdominal findings may not be made, and unnecessary surgical intervention may be carried out.

Summary

After the administration of antihypertensive drugs, a patient may develop symptoms and signs suggestive of an acute surgical abdomen. Within a period of a few months, we have seen four such patients.

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THROMBOPATHIC STATES

REPORT OF TWENTY-FOUR PATIENTS

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A group of 24 patients, all characterized by prolonged bleeding time with normal clotting time, were studied with respect to family history, personal history, bleeding time, prothrombin time, serum prothrombin, and thrombin generation. None showed abnormalities of clot retraction, platelet count, or morphology. No simple rule was found to explain the inheritance of this defect. The most important finding in the individual histories was excessive bleeding after tooth extractions. Classification on the basis of laboratory findings proved to be difficult, but the cases in general fitted von Willebrand's description of the thrombopathic state, and were shown to include variable abnormalities of labile prothrombin accelerator, platelet prothrombin accelerator, and platelet-co-thromboplastin. Adrenal steroids were used in 12 patients. This therapy was nonspecific, required two or three days to exert its characteristic action, but was effective in 10 patients.

Despite advances in diagnosis of hemorrhagic diseases, one is frequently faced with the clinical problem of the suspect bleeder. Routine bleeding and clotting times may be normal and thus mask a wide variety of potentially serious hemorrhagic states. These include especially the thrombopathic and related or synonomous syndromes of pseudohemophilia, familial hemorrhagic diathesis, thrombasthenia, and capillary bleeding; in addition, hemophilia and hypoprothrombinemia may be included. I consider a "mild" bleeder one who does not suffer severe spontaneous hemorrhage but who may have potentially serious post-traumatic hemorrhage. In the group to be discussed, prolonged bleeding time with normal clotting time is the common denominator.

The relative frequency of different types of hemorrhagic syndromes is well illustrated in the work of Cazal and Izarn,[1] who investigated the cause of hemorrhagic states in 273 patients. They were unable to demonstrate a defect in hemostasis in 149 (54%) and ascribed the bleeding to local vascular origin. The etiology of bleeding in the remaining 124 patients was as follows: in 46% it was attributed to thrombocytopenia, in 35% to increased vascular fragility, and in 19% to plasma coagulation defects. In only 11 of the 273 patients could it be classed as hemophilia. The present report is concerned with detection and treatment of relatively mild bleeders.

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Read before the 10th Clinical Meeting of the American Medical Association, Seattle, Nov. 29, 1956.

The role of the platelet in hemostasis is the resultant of several functions.[2] Complete agreement is by no means present; however, a working schema is shown in figure 1. The mechanism of platelet function in clot retraction is not well understood, but clot retraction does not occur in platelet-free

plasma or blood.[1] Physical factors, such as adhesiveness, undoubtedly play an important role in hemostasis, as indicated in the work of Wright [24] who demonstrated that heparin reduced adhesiveness of platelets while postsurgical and postpartum evidence for increased adhesiveness was shown. Evidence for a potent vasoconstrictor factor derived

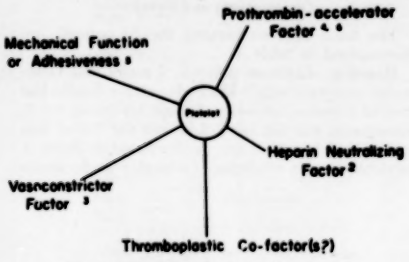

Fig. 1.—Role of platelet in clot formation.

from the platelet has been demonstrated in the work of Zuker.[25] Platelets have been shown to contain a prothrombin-accelerator factor which acts in a manner similar to AC-globulin [4] (conversion accelerator, V factor, labile factor). Perhaps the most important action of platelets has been shown to be that of a thromboplastic co-factor.[4] A heparin-neutralizing substance, a phosphatide, has been described [24] and a simple heparin-tolerance test devised for its detection. Clinical tests for evaluation of each factor are not available, although a prolonged bleeding time may be considered a crude measure of platelet dysfunction or absence.

Recent studies [5] have demonstrated that a normal clotting time does not exclude hemophilia. In a

TABLE 1.—*Differential Diagnosis of Antihemophilic Globulin (AHG), Plasma Thromboplastin Antecedent (PTA), and Plasma Thromboplastin Component (PTC) Deficiency**

	Corrected	
	Serum	$BaSO_4$-Treated Plasma
AHG deficiency	No	Yes
PTA deficiency	Yes	Yes
PTC deficiency	Yes	No

* Rosenthal.[5]

given patient with a slightly prolonged bleeding time and normal clotting time one must, therefore, specifically exclude deficiencies of antihemophilic globulin (AHG), plasma thromboplastin component (PTC), and plasma thromboplastin antecedent (PTA). The differential diagnosis of these groups by Rosenthal [5] is shown in table 1.

Bleeding times are usually normal in hypoprothrombinemic states; however, frequent exceptions have been noted so that the differential diagnosis of these states is included. Manipulations of the Quick one-stage prothrombin test have been used for clinical differentiation (table 2).

Methods

Patients were seen in the course of routine clinical hematological practice. Patients with frank hemophilia, severe hypoprothrombinemia, and

TABLE 2.—*Differential Diagnosis of Hypoprothrombinemic States*

	Prothrombin Time with Deficiency of		
	Prothrombin	Stable Factor	AC-Factor*
Plasma	Prolonged	Prolonged	Prolonged
Plasma plus stored plasma† (1:1)	Normal	Normal	Prolonged
Plasma plus stored serum (9:1)	Prolonged	Normal	Prolonged
Plasma plus prothrombin-free rabbit plasma (9:1)	Prolonged	Prolonged	Normal

* Deficiency of platelet AC-factor determined by addition of 0.1 volume washed platelets to test plasma uncorrected by above measures.
† 30-40-sec. Quick one-stage prothrombin time.

thrombocytopenia are excluded. Results in the study of 24 patients are reported. Table 3 highlights the methods used. All determinations were made on freshly drawn blood.

Bleeding time was determined in duplicate by using blood from both ears, and care was exercised to prevent massage of the ears or rubbing of the skin with the blotter paper. In nearly all cases the bleeding time determination was repeated on several occasions, days to weeks apart. Prothrombin-free rabbit plasma was used in prothrombin

TABLE 3.—*Methods Used in Evaluation of Hemorrhagic Tendency*

Determination	Method	Normal*
Bleeding time	Duke[6]	<4.5 min.
Prothrombin consumption	Quick[4]	Serum prothrombin 15% or less at 1 hr.
Thrombin generation	Pitney and Dacie[7]	1 unit thrombin/ml. of thrombin-fibrinogen mixture clots in 18-30 sec. at 37° C. Normal with 4-8 units of thrombin at 4 min.
Platelets	Dameshek[7a]	200,000-300,000/cu.mm.
Clotting time and retraction	Lee and White[6]	5-15 min. with retraction complete within 1 hr.
Heparin-neutralizing substance	Van Creveld[2a]	Tube no. 1 (1/ug. heparin clots 20-30 min.) Tube no. 2 (2/ug. heparin clots 25-40 min.) Tube no. 3 (4/ug. heparin clots 40-60 min.)
Prothrombin time	Quick[4]	13-13.5 sec. = 100%

* Refers to normal values obtained in our laboratory.

consumption tests. Screening for fibrinolysin was evaluated by observation of clot breakdown in the tubes during the performance of the clotting time and retraction tests. Lysis of the clot in less than 45 minutes was considered abnormal.

Platelet suspensions were freshly made daily. Blood was collected from both the patient and normal control subject on each determination, with use of silicone technique without anticoagulants. A pellet of platelets was obtained from 20 ml. of blood by centrifugation in an ice bath, by washing

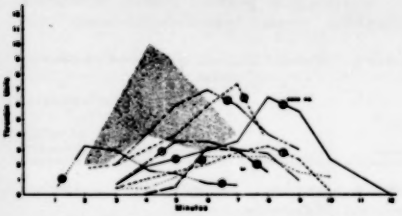

Fig. 2.—Abnormal thrombin generation in thrombopathic states (shaded area is normal range).

three times in isotonic sodium chloride solution, and then by suspension in 1.5 ml. of isotonic sodium chloride solution. One part of normal platelet suspension was added to three parts of fresh blood in the experiments evaluated by prothrombin consumption testing. One-tenth volume of platelet material was added to decalcified plasma in those cases evaluated by the thrombin generation test. The effects of the platelet additions were evaluated in normal persons, in patients with hemophilia (AHC deficiency), and in controls with thrombocytopenia, as well as in patients studied.

The thrombin generation test, as performed on recalcified plasma,[7] was used as a further check on the prothrombin consumption test. A purified form

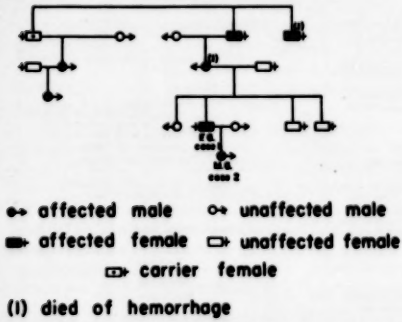

Fig. 3 (table 4, family no. 1).—Family tree for two patients with thrombopathy (von Willebrand type).

of human fibrinogen was used in this test. Samples from recalcified plasma are added at one-minute intervals to tubes containing standard fibrinogen solution. The concentration of thrombin is inferred from the rate of fibrinogen clotting. A standard curve was prepared with use of known solutions of thrombin (Thrombin topical) so that from any sample the time of the clotting could be converted to thrombin units. The normal curve used in this study, (fig. 2), based on results in 30 normal persons tested, compares favorably with that reported by other authors.[7]

Observations and Results

The basic data concerning the 24 patients are summarized in table 4.

Heredity.—Eighteen patients, 7 males and 11 females, representing 11 kindreds, gave a family history of bleeding tendency. Except for family no. 2, inheritance was not sex-linked and the defect was transmitted by either sex, as illustrated in figure 3. In contrast, the sex-linked (x-borne) mode of in-

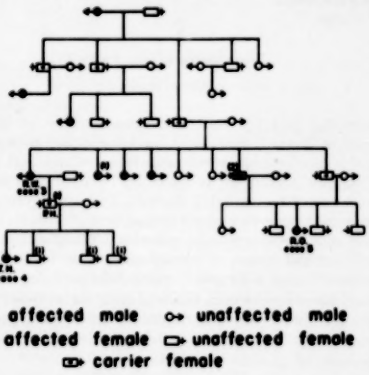

Fig. 4 (table 4, family no. 2).—Family tree for three patients with unclassified thrombopathy.

heritance predominated in this family, as illustrated in figure 4. In the remaining six patients there was no known family history of bleeding tendency.

Effect of Bleeding on General Health, and Deaths.—The patients in cases 1, 2, 4, 6, 7, 9, 14, and 18 presented variable stages of chronic ill-health characterized by fatigue, muddy complexion, chronic anemia, repeated mouth infection, and dental caries. The patient in case 18, a reliable observer, noted that fatigue would be followed by bleeding from the nose and gingiva and that remissions were accompanied by a sense of increased strength and well-being. Menorrhagia was almost constant for the first four years after onset of menses in the patient in case 14. Fear of postextraction hemorrhage occasionally caused undue delay in seeking adequate dental care. Seven deaths were attributed to bleeding tendency in the family histories. One additional death occurred in this series.

	Case No., Age, Yr., and Sex	Family History	Personal History	Bleeding Time, Min., Sec. (Normal, 2-4 Min.)	Prothrombin Time (Normal, 100%)	Serum Prothrombin (Normal, <15%)	Thrombin Generation (Normal, 4-8 Units at 4 Min.)	Remarks*	Classification
Family No. 1	1—22, F	Father bruised easily, had frequent epistaxis, and died after tooth extraction; paternal aunt died of postoperative hemorrhage; mother of patient in case 2	Frequent epistaxis, menorrhagia, easy bruising, chronic ill-health due to blood loss	5 24 6 11 14	100 40 100 100 57	16 14 14 16 35	...	Therapy with protamine sulfate, 150 mg., intravenously, shortened bleeding time from 14 and 16 min. to 8 min., 30 sec. on two trials; 250 ml. in normal saline also reduced bleeding time from 11 min., 30 sec. to 7 min., 30 sec.; AC-globulin, vitamin deficiency, and increased heparin excluded	Thrombopathy (von Willebrand type)
	2—4, M	See case 1; 7 known bleeders in family; simple dominant pattern of inheritance	Bruises easily, epistaxis, chronic ill-health, nearly exsanguinated after minor cut on tongue	4 20 5	80 100	35 57	...	Therapy with protamine sulfate, 100 mg., and blood given intravenously without benefit in control of tongue laceration; controlled by suturing	Thrombopathy (von Willebrand type)
Family No. 2	3—53, M	Three brothers and (questionably) one sister bruise easily; frequent epistaxis, and postextraction bleeding; cousin had chronic ill-health due to repeated hemorrhages; 10 known bleeders in family; sex-linked recessive pattern; grandfather of patient in case 4	Epistaxis, frequent bruising, postextraction hemorrhage, and rectal bleeding since childhood; bleeding frequently delayed 7-14 days after surgery	5,30 8,30 2 3 4 7	100 90 100 100	4 1 4 	Delayed	Fresh whole blood given without benefit; severe total postextration hemorrhage stopped on 3 trials with slow intravenous drip of 25 mg. ACTH in 1,000 cc. dextrose solution	Thrombopathy, unclassified
	4—7, M	Two sisters, free from bleeding tendency, have normal serum prothrombin content; 2 brothers bruise easily, have delayed thrombin generation	Severe post-tonsillectomy and postextraction hemorrhage, gastrointestinal and renal hemorrhages, bruises easily, epistaxis; chronic ill-health	8 7,15 5	100 100 ...	45 29(a) 29(b)	Delayed	(a) Corrected, in vitro by platelets from grandfather (case 3); normal control (b) 300 cc. fresh blood in silicone, yielding 300,000 platelets per cu. mm., did not correct prothrombin consumption	Thrombopathy, unclassified
	5—21, M	Nephew of patient in case 3; mother free from bleeding trait; family history as for cases 3 and 4; 2 sisters free from bleeding tendency	First bleeding, age 1 yr., from minor cut; hospitalized, age 4, with cut lip; transfusion gave questionable help; at age 5, cut lip and bled one week; at age 11, cut head, bandaged with pressure for 2 mo. with intermittent bleeding and massive hematoma; bled 3 wk. from minor palatal scratch; epistaxis	2,35 2,40 3	100 100	3 3	Delayed	Test for heparin neutralization normal; thrombin generation corrected with platelets; partial correction with normal and PTC-deficient plasma	Thrombopathy, unclassified
Family No. 3	6—20, F	Paternal grandmother, father, and two uncles were bleeders; father and his brother died of gastrointestinal hemorrhage; mother of patient in case 7	Epistaxis, postextraction, and postpartum hemorrhages required transfusions	>10	100	...	Normal	...	Thrombopathy (von Willebrand type)
	7—1½, F	See case 6	Repeated severe epistaxis; bleeding from minor cuts	8 >20 20	100	...	Normal	...	Thrombopathy (von Willebrand type)
Family No. 4	8—58, M	Mother had frequent epistaxis, menorrhagia, bruising, and postextraction bleeding and died of gastrointestinal hemorrhages; brother died of gastrointestinal hemorrhages; one of the latter's twins (son) is a bleeder, and the twin daughter is normal; father of patient in case 9	Frequent nosebleeds during childhood required packing; minor cuts bled excessively until approximately age 30 yr.; postextraction bleeding, age 14; massive gastrointestinal hemorrhages, age 22, with negative x-rays, massive gastrointestinal hemorrhages, age 50; mild bleeding tendency thereafter	7,5 5,55	100	15	...	...	Thrombopathy (von Willebrand type)
	9—35, M	Son of patient in case 8; two daughters bruise constantly	Frequent nosebleeds requiring packing; excessive postextraction bleeding during childhood; tarry stools; onset age 35, intermittent to date and requiring many transfusions; chronic invalid state due to blood loss	20 9 20 10(a) 5(b) >30(c) 12(c) >40	78 60 90 80(a) 80(b) 60(c) 97(d) 79	2	Delayed	(a) After 500 cc. and (b) after 1,500 cc. fresh whole blood transfusion with glass equipment; (c) before and (d) after 500 cc. whole polycythemic blood given intravenously in silicone equipment; test for heparin neutralization normal; ACTH and cortisone therapy given without benefit	Thrombopathy (von Willebrand type) with platelet and plasma AC-globulin deficiency
	10—5, F	Daughter of patient in case 9; see cases 8 and 9	Age 2, lip cut with mild bleeding, sutured for control; bruises on body constantly	>30 >30	100 100 90	29 5	Normal	Heparin neutralization probably normal	Thrombopathy (von Willebrand type)

* Clotting times, platelet counts and morphology, and clot retractions were normal throughout.

TABLE 4.—*Summary of Findings in Twenty-four Patients with Thrombopathic States* (Continued)

Case No., Age, Yr., and Sex	Family History	Personal History	Bleeding Time, Min., Sec. (Normal, 2-4 Min.)	Prothrombin Time (Normal, 100%)	Serum Prothrombin (Normal, <15%)	Thrombin Generation (Normal, 4-8 Units at 4 Min.)	Remarks*	Classification
11—28, F	Sister, mother, and maternal great-grandmother had excessive postpartum hemorrhages, frequently delayed	Post-tonsillectomy and postpartum hemorrhages, requiring transfusions	4 9,30	100	20 (control, 18% on this occasion)	...	Therapy with vasopressin (Pitressin) by slow intravenous drip, prolonged, claimed to have helped postpartum hemorrhages	Thrombopathy (von Willebrand type)
12—32, F	Father had frequent epistaxis; additional history unavailable	Epistaxis requiring frequent packing; bleeding for 1 wk. after submucus resection and after tonsillectomy; transfusions ineffective; severe menorrhagia	10 11 5,55 7,10	63 89 100 79	... ...	Delayed Delayed	Bleeding time and impaired thrombin generation worsened postoperatively (dilatation and curettage) even though 1 unit fresh blood given in plastic; ACTH and prednisone (Meticorten) effective in post-dilatation and curettage bleeding and for menorrhagia	Thrombopathy (von Willebrand type)
13—48, F	Four known bleeders; 6 with equivocal history of bleeding tendency, either sex affected; bleeding, postextraction and post-tonsillectomy; menorrhagia noted	Spontaneous gingival bleeding; epistaxis; mild left hip hemarthrosis; bruises easily	5 11 9,30 4,15	100 100 100	 5	Normal	Heparin neutralization normal; excellent control of extractions with cortisone and local therapy as compared with prior extractions	Thrombopathy (von Willebrand type)
14—30, F	Uncertain; mother had menorrhagia	Long history of easy bruising and epistaxis as a child; nearly constant menstrual bleeding for 4 yr. after onset, now very heavy but lasts 5-6 days; bled 4 days postextraction; oophorectomy, however, without serious bleeding	6,30 8,30 15	... 100 90	16 ... 16	...	...	Thrombopathy (von Willebrand type)
15—16, F	Mother had menorrhagia and epistaxis and died after delivery due to postpartum hemorrhage; maternal aunt bruises easily	Episodic gingival and nasal hemorrhages, frequently lasting for months	3,30 4,26 2,45 10 10	100 100	18	Normal	Gingival bleeding nearly constant during night; prednisone used with complete control	Thrombopathy (von Willebrand type)
Family No. 5 16—33, F	Father and paternal aunt with epistaxis and postextraction bleeding; daughter of patient in case 17	Childhood epistaxis; severe menorrhagia, postpartum and postextraction hemorrhage, frequently delayed; spontaneous bleeding from migratory telangiectasia	7,5 4,50 5,35	100	...	Normal	ACTH and prednisone effective; extractions without abnormal bleeding	Thrombopathy (von Willebrand type)
17—12, F	See case 16; one brother normal; one brother has mild bleeding tendency	Bruises easily; prolonged bleeding from cuts; severe post-tonsillectomy hemorrhage	>22	61	...	Normal	Prothrombin corrected in vivo with prothrombin-free rabbit plasma or fresh platelet-free plasma; uninfluenced by serum	Thrombopathy (von Willebrand type) with labile prothrombin deficiency
18—52, F	Negative	Spontaneous onset of bleeding in 1945 after 20 injections of gold for arthritis, with no known toxic reaction; appendectomy, 1926, and extractions up to 1944 without evidence of hemorrhage; chronic anemia, intermittent severe gingival bleeding, and prolonged episodes of epistaxis	2,30 >25 7 8,30 7,0 >25	100 100 100 100 100 100	16 35(a) 8 2 31(b)	Delayed	(a) Histamine (as below, case 19) to tolerance dose of 0.69 mg. did not correct serum prothrombin or shorten bleeding time; protamine sulfate was of no value on 2 trials (b) 500 cc. of polycythemic blood given intravenously in plastic reduced serum prothrombin to 11%; hydrocortisone stopped bleeding	Acquired thrombopathy
19—45, M	Negative	Spontaneous onset while receiving gold therapy for rheumatoid arthritis; also developed toxic nephrosis; bleeding at sites of needle punctures; easy bruising; onset on 1/7/53 and persisted to 1/5/54	13 ... ... 12,30(a) 10 11 4 3,5	100 ... ... 75(a) 97 72 100 100 100	... ... 45 58 40(a) 39 37 29 90 2	...	(a) When 1.82 mg. histamine diphosphate was given intravenously plasma prothrombin of 75% and serum prothrombin of 40% both converted to normal; first normal serum prothrombin on 12/30/54, after prolonged therapy with ACTH	Acquired thrombopathy
20—30, F	Negative	Menorrhagia and easy bruising for many years	5 5,30 92 4 10 ...	72 70 80 75 80 77 and 68(a)	11	...	Vitamin K_1 oxide given without improvement (a) Prothrombine time before and after extraction respectively	Acquired thrombopathy with mild plasma AC-globulin deficiency
21—25, M	Negative	Tonsillectomy, age 4, and orthopedic surgery, age 10, carried out without difficulty; 1st noted postextraction oozing for 1 wk., age 17; referred because of 11-day episode of postextraction bleeding	9,35 12	50 50 65	...	Normal	Liver profile and plasma electrophoresis normal	Acquired thrombopathy with AC-globulin deficiency

* Clotting times, platelet counts and morphology, and clot retractions were normal throughout.

TABLE 4.—*Summary of Findings in Twenty-four Patients with Thrombopathic States* (Continued)

Case No., Age, Yr., and Sex	Family History	Personal History	Bleeding Time, Min., Sec. (Normal, 2-4 Min.)	Prothrombin Time (Normal, 100%)	Serum Prothrombin (Normal, <15%)	Thrombin Generation (Normal, 4-8 Units at 4 Min.)	Remarks*	Classification
22—61, F	Unknown	Bled excessively after extraction; history of easy bleeding noted; fatal hemorrhage after thyroidectomy	18 12	52 47	2 2	...	ACTH given at time of first set of data, and second data taken 2¼ hr. later; patient died suddenly; autopsy showed deep neck hemorrhages as cause of death	Acquired thrombopathy (?) with hypoprothrombinemia (type undetermined)
23—18, M	Questionable maternal history of menorrhagia and epistaxis	Frequent and severe epistaxis for 2 yr.; extractions, 2/31 and 10/32 without difficulty; mild to severe delayed (5 days) postextraction bleeding since 1953	5 11 4 3 3	100 78 100 100 100	... 11 32 24 25	Delayed	Tooth extraction, with ACTH, thrombin-gelfoam pack, and sutured, successful; thrombin generation delay corrected with platelets or serum	Thrombopathy (von Willebrand type)
24—5, M	Negative	Almost daily nosebleeding and bruising for past 8 mo.; cerebral palsy with mental deficiency	9,5 7,15(a)	100 100(a)	41 26(a)	Abnormal	(a) Readings 18 hr. after receiving ACTH, 25 mg., and hydrocortisone, 60 mg.; stopped bleeding in 24 hr. and remained free for 2 wk.; intermittent cortisone therapy has controlled bleeding for 2 yr.; vitamin K and large doses of ascorbic acid for 6 wk. previously of no value	Thrombopathy (von Willebrand type)

*Clotting times, platelet counts and morphology, and clot retractions were normal throughout.

Bleeding Time.—The variability of bleeding time is graphically shown in figure 5. The volume of bleeding, as seen on the filter paper, was frequently excessive, apart from the time factor itself.

Clotting Time, Clot Retraction, and Platelets.—We found no abnormalities in clotting time, retraction, platelet count, or morphology.

Plasma Prothrombin.—Plasma prothrombin studies are shown in tables 4 and 5. Abnormal one-stage prothrombin times were recorded in the plasma of six patients. Patients in cases 9, 12, 17, 20, and 21 are shown to have AC-globulin deficiencies (table 5).

Prothrombin Consumption.—Variability of the serum prothrombin, which ranged from normal to abnormal, is shown in tables 4 and 6. In members of the same family who were bleeders, it varied so

TABLE 5.—*Differential Diagnosis of Prolonged One-Stage Prothrombin Content*

Case No.	One-Stage Prothrombin Range, %	Effect of Added Fractions on One-Stage Prothrombin Test, %: Prothrombin-free Plasma* (9:1)†	Aged Serum (9:1)	Fresh Plasma (1:1)	Stored Plasma (1:1)	Platelets (9:1)
1.....	40-100	...	...	...	...	...
12.....	63-100	100	63-100	...	63-100	63-100
9.....	60-80	100	60-80	...	50	100
17.....	61	100	60	100	...	60
20.....	63-80	100	85	100	40	70
21.....	50-65	100	40	100	40	...
22.....	47-52	55	...	...	...	...

* Prothrombin-free rabbit plasma.
† Volume patient plasma : volume added substance.

that while one usually had normal serum prothrombin (as in cases 1, 3, and 5) another might be abnormal (as in cases 2 and 4).

Freshly washed human platelets corrected the serum prothrombin time in cases 4, 18, 19, and 24, but not in case 23. Control plasma from a patient with idiopathic thrombocytopenic purpura with platelets reduced to 8,000 to 10,000 per cubic millimeter was shown to have a serum prothrombin time of 12 seconds which could be corrected to 120 seconds (less than 1%) with comparable platelet additions.

TABLE 6.—*Range of Serum Prothrombin in Thrombopathic States with Abnormal Values and Effect of Adding Fresh Normal Platelets*

Case No.	Serum Prothrombin Range, % (Normal to 15%)	Serum Prothrombin After Adding Platelets (1 Vol. Platelets:3 Vol. Whole Blood)
1.....	14-35	...
2.....	35-57	...
4.....	29-45	4-7
10.....	5-20	...
18.....	2-35	4
19.....	59-90	2
23.....	11-32	20*
24.....	36-41	3

* Not corrected.

Thrombin Generation.—The results for family no. 2 will be considered first. A normal thrombin unit value was obtained but was delayed two to three minutes in the test on unmodified blood and four to six minutes on the test on recalcified plasma.[7] Figure 2 illustrates the skew to the right (delayed generation) of the thrombin generation curves in the above kindred and also in cases 9, 12, 18, 23, and 24. In all except the patient in case 24, platelets corrected the abnormal thrombin generation (table 7). Plasma from this patient was unusual in that thrombin generation began prematurely and abruptly declined. In case 23, serum (PTC and PTA) partially corrected the delay while platelets alone produced complete correction. Because of the sex-linked recessive pattern in family no. 2, numerous tests (table 7) were conducted to exclude AHG and PTC deficiency, including the use of patient

plasmas deficient in these factors. Equivocal results in cases 3 and 4, suggesting mild associated AHG deficiency, were shown (table 7). Platelets from patients in cases 4 and 18, when added to platelet-

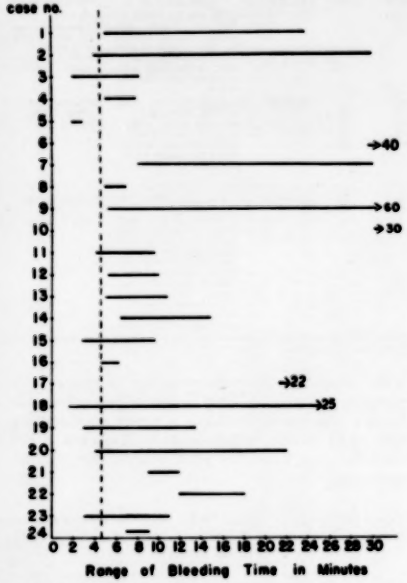

Fig. 5.—Variability of bleeding time in patients with thrombopathy.

free normal plasma, resulted in normal thrombin generation, while those from the patient in case 12 were ineffective.

Therapeutic Trials.—In 1950, a female patient was seen for serious postextraction hemorrhage despite transfusion with 12 pints of fresh whole blood. Hemorrhage was dramatically controlled with 150 mg. of protamine sulfate in 1,000 cc. of dextrose solution given intravenously. Bleeding was stopped within a period of 10 to 15 minutes on three occasions, and finally a prolonged 24-hour intravenous drip completely controlled bleeding. With that experience, this drug was tried in several other patients. However, none responded (cases 1, 2, and 18).

Use of silicone-treated transfusion equipment was begun, and results have been encouraging. The patient in case 9 had received 24 units of whole blood, much of it fresh, without control of gastrointestinal hemorrhage. The first unit of polycythemic silicone-protected blood increased the plasma one-stage prothrombin time from 65% to 95% of normal and reduced the bleeding time from greater than 30 minutes to 12 with marked reduction in intensity of bleeding. Subsequently, each of six episodes of massive gastrointestinal hemorrhage have been controlled by a routine of 2 units of fresh blood in plastic bags every 12 hours for 48 to 72 hours. Bleeding and prothrombin times have become normal at the close of this period.

I wished to observe the effect of intravenously administered histamine. Eosinophil counts taken prior to the infusion revealed only 2 per cubic millimeter in each of two cases, as compared with a normal control patient. Histamine diphosphate (0.69 mg.) given intravenously to the patient in case 18 to a point of severe headache had no effect on the impaired serum prothrombin content of 35%. Her control bleeding time was over 25 minutes and was 20 minutes after the infusion. Platelet clumping was noted to be present before and after the experiment. However, in the patient in case 19, after 1.82 mg. of histamine diphosphate was given to a point of asthmatic symptoms, the plasma prothrombin content of 75% was corrected to 100% and serum prothrombin of 40% was corrected to 17%. Bleeding time of 12.5 minutes was reduced to 6 minutes. Platelet clumping, which was absent before the infusion, was present after the infusion.

The use of steroids was begun in January, 1953, and my experience with patients having thrombopathic states is summarized in table 8. Two failures were noted among the 11 patients treated. The patient in case 9 has been completely refractory to intensive steroid therapy. That in case 22 died in 2½ hours, so that the effects of prolonged therapy were unobtainable. An additional patient with thrombopathic state not included in this report was prepared for removal of a cataract with use of steroids, and a completely normal course resulted. The chief hazard of steroid therapy is the develop-

Table 7.—*Differential Diagnosis of Cases with Delayed Thrombin Generation**

Case No.	Platelets	Normal Plasma	$BaSO_4$ Plasma	48-Hr. Serum	PTC-Deficient Plasma
3	C	PC	PC	UC	PC
4	C	UC	UC	UC	UC
5	C	PC	UC	UC	PC
9	C	UC	UC	UC	UC
12	C	PC	UC	UC	...
18	C	UC	UC	UC	UC
23	C	UC	UC	PC	...
24	PC	UC	UC	UC	...

* C, corrected; PC, partially corrected; UC, uncorrected.

ment of thrombophlebitis. This occurred in cases 3 and 13. Pulmonary embolism occurred in case 13 and in two other cases not included in this report.

Comment

The cases described fit the von Willebrand-Jürgens description of thrombopathic state and showed marked variability in bleeding time and hemor-

rhagic manifestation. However, the sole finding of a prolonged bleeding time is not proof that surgery will be complicated. Major surgery may be less of a problem than a simple tooth extraction. I have observed not infrequently that minor surgery, involving teeth, eyes, tonsils, and skin, may lead to excessive bleeding with poor wound healing not associated with major procedures in the same patient.

Prolonged bleeding times are usually not seen with prothrombin or accelerator defects, although exceptions have been recorded.[8] Several of my patients with lowered prothrombin times had prolonged bleeding times even though the prothrombin defect was not of the same origin in each. Prolonged bleeding time does not exclude hemophilia. Consequently, a prolonged bleeding time does not identify the disease state, and more detailed studies are required. The defects in prothrombin times rarely approached a level of 20 seconds (approximately 40%), which Quick has listed as critical.[8a] Even so, the bleeding tendency seen in this hypoprothrombinemic group was at times severe, and it was fatal in one. In these instances more than one factor may be operative. This is illustrated by Owren's case of a 5-year-old boy with a Quick one-stage prothrombin time of 15 to 16 seconds but only a 35% content of proaccelerin.[9]

A normal clotting time does not exclude a hemorrhagic diathesis in which a defect in thromboplastin formation may actually exist. Thus, 40% of our patients were found to have defects in thromboplastin formation. Jürgens, as quoted by Wintrobe,[10] also observed delayed prothrombin consumption in cases of classic von Willebrand hemorrhagic diathesis. The defect in thromboplastin formation in our patients was frequently corrected by washed platelets. Three patients with excellent prothrombin consumption tests had abnormal thrombin generation tests which were corrected with platelets. Washed normal platelets did not improve thrombin generation in proved AHG deficiency. Patients with PTA deficiency may resemble patients with thrombopathic states. PTA factor, however, is readily supplied by stored blood.

The experimental work of Jürgens [11] indicated that agglutination of platelets was markedly promoted by histamine, which leads to localized fibrin flocculation. Sanford and others [12] demonstrated the absence of an in vitro action of histamine on dog or human platelets, but an increased clumping and disintegration followed intravenous injection which was directly related to the degree of reaction experienced by the patient. Heparin therapy did not prevent this action. Two patients with impaired prothrombin consumption correctible with platelets were given histamine intravenously. The results in one correlate with the concept of a thrombasthenia which was corrected through the action of histamine on platelets possibly mediated as above.

The in vitro effects of added platelets suggested that transfusions with use of silicone-treated or plastic equipment might be more effective than ordinary glass-contained blood, and this appeared to be true. The possible trigger role [13] of the platelet, which may influence directly or indirectly the activation of AC-factor and stable prothrombin accelerators, suggests an additional reason to use every measure to ensure maximum platelet delivery.

TABLE 8.—*Results of Steroid Therapy in Thrombopathic States*

Case No.	Status	Steroid	Notes and Results
3	Total extraction, severe bleeding	ACTH by continuous intravenous drip	Three units fresh blood and protamine sulfate given without effect; bleeding stopped in 2 hr.; trial repeated 3 times
4	Tooth extraction	Prednisone, 20 mg./day, begun 48 hr. preoperatively	Oozed for 1 mo. with previous extractions; normal without excessive bleeding this occasion
9	Repeated massive gastrointestinal hemorrhages	ACTH by continuous intravenous drip and orally given cortisone for 1 and 2 wk. respectively	Prolonged bleeding time and melena unimproved
12	1. Dilatation and curettage 2. Menorrhagia	1. ACTH by continuous intravenous drip 2. Prednisone, 15-20 mg./day	1. Normal course after dilatation and curettage 2. Flooding menses prevented by treatment, beginning 48 hr. premenstrually; Bleeding returned with omission of drug
13	1. Tooth extractions. 2. Cholecystectomy with common duct stone	1. Hydrocortisone, 60 mg./day 48 hr. preoperatively 2. Hydrocortisone, 60 mg./day; intravenous drip, ACTH	1. Uneventful compared with prior experiences according to dentist and patient 2. No hemorrhage; severe thrombophlebitis with pulmonary embolus
15	1. Nearly constant gingival oozing, especially nocturnal 2. Delivery	1. Prednisone, 20 mg./day 2. Intramuscularly given hydrocortisone and continuously given ACTH	1. Complete cessation in 36 hr. 2. Post partum immediate hemorrhage; given 1,000 ml. of blood; further hemorrhage controlled with continuously given ACTH
16	Multiple extractions	ACTH given by continuous intravenous drip, followed by prednisone	Normal postoperative course
18	1. Gingival hemorrhage 2. Gingival and nasal bleeding	1. ACTH by continuous intravenous drip for 24 hr. 2. Hydrocortisone 60 mg./day; later prednisone, 15-20 mg./day	1. Bleeding unaffected; transfusions also given 2. Bleeding stopped in 3 days and has been controlled by intermittent therapy for 1½ yr.; many transfusions previously
22	Post-thyroidectomy hemorrhage	ACTH by continuous intravenous drip	Progressive oozing and death due to hemorrhage 2¾ hr. after giving ACTH
23	Tooth extraction	Hydrocortisone, 60 mg./day	Normal postextraction course, as compared with previous experiences of 3-7 days bleeding
24	Repeated epistaxis and bruises	Prednisone, 15-20 mg./day for 10-20 days	Bleeding controlled; courses of treatment required every 2-3 mo.; maintained well for 2 yr. to date

My experiences from 1949 to the time of writing in treating patients having various types of purpura with ACTH indicated its effectiveness in the frequent control of purpura without necessarily affecting the platelet count. Others have recorded similar observations.[14] The exact mechanism of control of capillary hemorrhage is not clear. Cosgriff and others [15] demonstrated hypercoagulability of the blood, lasting 7-15 days after treatment, in association with ACTH and cortisone therapy. The improvement in my patients did not always correlate with correction of bleeding time or prothrombin consumption. Jacobson [14] demonstrated a reduction in bleeding time after the use of cortisone and ACTH in patients with pseudohemophilia. I am continuing to use prednisone (Meticorten) or hydrocortisone (Compound F) in the preoperative preparation of these patients, a number of whom are not included in this report. I have recommended the following course in preextraction cases: preparation of precasted bite-plates for extensive extractions, preparation with steroids (60-120 mg. of hydrocortisone or 30 mg. of prednisone daily) for 48 hours preoperatively, packing with absorbable surgical sponge (Gelfoam) and topical thrombin, and suturing. Steroid administration is gradually reduced over one week. Bleeding has been reduced, as compared with patients' previous experiences. This far, the mechanism appears nonspecific. Thrombophlebitis is the chief complication and appears to be more severe than in normal states.

Theoretically, protamine sulfate would be specific in rare instances where increase in heparin-like substance or defect of platelet factor 3 [25] exists. My results suggest that a test dose should be sufficient, with hemorrhage promptly arrested if the drug is effective. Intravenously given conjugated estrogenic substances (Premarin) has been used in scattered cases and is most helpful when labile prothrombin defect coexists. Controlled experiments during serious bleeding are difficult to set up; further, variability from episode to episode is most difficult to correlate.

Summary

A clinical approach to the evaluation of the mild or questionable bleeder has been studied in 24 patients with thrombopathic states. A searching history is the best screening test. A positive family history was obtained in two-thirds of the patients and was usually manifested as a simple dominant trait. In one family a recessive (x-borne) type of inheritance was demonstrated in which a platelet-plasma thrombopathic state was present associated with mild hemophilia.

A variably prolonged bleeding time was the most frequent laboratory finding, whereas a history of excessive bleeding after tooth extraction was the most important anamnestic observation. Other important symptoms were easy bruisability, epistaxis, gingival bleeding, and menorrhagia. Purpura was absent. Approximately one-half of the patients at some time had abnormal prothrombin consumption and/or thrombin generation.

A mild to moderate reduction in the one-stage prothrombin content, due to AC globulin (conversion accelerator, V factor, labile factor) deficiency, was noted in about one-fourth of the patients and may be only one facet of a potentially serious defect in hemostasis. One post-thyroidectomy fatality occurred in this group.

Classification is difficult and frequently artificial since the variation in findings among bleeders in a single kindred group could frequently lead to multiple diagnoses. The cases in general fit the so-called thrombopathic state of von Willebrand and include variable abnormalities of labile prothrombin accelerator, platelet prothrombin accelerator, and platelet co-thromboplastin defect. Three bleeders, not reported on, had normal clotting times and slightly prolonged bleeding times; two were shown to have antihemophilic globulin (AHG) deficiency, and one had plasma thromboplastin component (PTC) deficiency. From the standpoint of management it is important to evaluate the individual and define the defect in terms of a therapeutic approach.

Therapy is often difficult, and whole blood transfusions are usually without direct benefit. The use of silicone-treated and plastic-protected blood is of aid. Adrenal steroids were used in 12 patients and were effective in 10. The effect is nonspecific and may require two to three days to become manifest. Thrombophlebitis is a serious complication of this therapy. Control of bleeding persisted for several weeks after stopping administration of the drug, so that intermittent therapy provided excellent control of chronic epistaxis, menorrhagia, and gingival bleeding. Combination of regularly spaced transfusions of fresh blood or plasma in plastic bags, adequate local measures, plus steroids is optimal treatment of the thrombopathic hemorrhagic state. Therapy with conjugated estrogenic substances (Premarin) is indicated when deficiency of labile prothrombin accelerator is present.

Addendum

Since this paper was read on Nov. 29, 1956, an additional member of family no. 2 has been studied and is shown to have a definite mild antihemophilic globulin deficiency, demonstrating in this family a combination of thrombopathic states with hemophilia.

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HORMONES, VIRUSES AND CANCER.—The administration or withdrawal of hormones can stimulate or control the growth of certain cancers of the human breast, prostate, and thyroid. In animals, too, it has been shown that the growth of cancers can be stimulated by disturbing endocrine balances and that often these cancers can be controlled by restoring normal endocrine balance. Prolonged and excessive stimulation of a target organ by a trophic hormone first causes hyperplasia of the animal's target organ, then formation of benign nodules, and then cancers. At first these cancers may be dependent for their continued growth on the persistence of the endocrine imbalance that produced them but, finally, if the imbalance persists, autonomous cancers develop and can grow in animals in which there is no longer any endocrine imbalance; for example, the production of cancer of the thyroid by feeding thiouracil. . . . Excessive stimulation by a trophic hormone stimulates a cell to divide but, before it divides, the cell must reproduce its normal quota of nucleoprotein. A stimulated cell's avidity for nucleoprotein may become so great that the cell not only reproduces its own nucleoproteins as fast as it can but also gobbles up any virus-like particles of nucleoprotein with which it comes in contact. These particles, incorporated into the chromatin of the cell, might disturb the cell's genetic balance. Thus exposure of a trophin-stimulated cell to a virus might hasten the development of the nuclear changes that lead to autonomy of growth. Viruses may be part of the material out of which cancers are built.—George Crile Jr., M.D., A Speculative Review of the Role of Endocrine Imbalances in the Genesis of Certain Cancers and Degenerative Diseases, *Journal of the National Cancer Institute*, February, 1958.

MITRAL INSUFFICIENCY—ITS QUANTITATION BY CARDIAC VENTRICULOGRAPHY

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Thirty-two patients had cardiac ventriculographic study for demonstration of the presence and degree or absence of mitral regurgitation. Four patients showed no atrial opacification, and at surgery these four were proved to have pure mitral stenosis without any mitral regurgitation. Surgical treatment of mitral stenosis demands an effective procedure for the preoperative assessment of the presence and degree or absence of mitral regurgitation. Any degree of regurgitant opacification of the left atrium during cardiac ventriculographic study is considered significant of mitral valvular insufficiency. Cardiac ventriculography does not lend itself to exactitude of interpretation but rather to estimation only of the amount of regurgitant opaque substance issuing through the mitral valve and the quantitation of mitral insufficiency. It also appears to be a reasonably efficient procedure for evaluating the effectiveness of surgical procedures designed for the correction of mitral insufficiency.

The need for an accurate clinical method for quantitation of mitral regurgitation has long been recognized and has been an elusive problem. Since the advent of surgical treatment of mitral stenosis,[1] the importance of an effective procedure for the preoperative assessment of the presence and degree or absence of mitral regurgitation has become manifest. Anatomic changes present in the mitral valve leaflets inducing regurgitation may preclude successful mitral valvular mobilization. Since in our institution mitral stenosis is now approached surgically through an incision on the right side and mitral insufficiency through an incision on the left side, it has become doubly important to establish with certainty the preoperative diagnosis.[2]

Although catheterization of the left side of the heart has proved of inestimable value in the diagnosis of mitral and aortic stenosis, it has not been adequate in the evaluation of mitral regurgitation.[3] Cardiac ventriculography has provided a more accurate means of objective assessment of the presence and degree of mitral valvular insufficiency. Cardiac ventriculography is also an effective means for evaluating the efficaciousness of surgical procedures designed for correction of mitral insufficiency.

Smith, Cregg, and Klassen [4] have discussed the diagnosis of mitral regurgitation by cardioangiography. These authors employ the term "cardioangiography," while we prefer the term "cardiac ventriculography" for the procedure of direct transthoracic needle puncture opacification of the ventricles of the heart. Both terms refer to the same procedure.

Smith and his associates [4] studied 20 patients with consideration of the state of competency of the mitral valve, 10 patients presenting evidence of a competent mitral valve and 10 showing varying degrees of mitral regurgitation. Anatomic evidence of the status of the mitral valve was available in 6 of the 20 cases studied. These authors report essential agreement relative to the competency of the mitral valve between cardioangiographic findings and subsequent anatomic findings at surgery or autopsy in five of these six patients.

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The purpose of the present report is to describe experience in the estimation of mitral insufficiency by cardiac ventriculography and to present observations as to the correlation of the ventriculographic quantitation of mitral insufficiency with the operative findings at the time of surgical exploration of the mitral valve.

Methods and Materials

Our technique of cardiac ventricular puncture and radiologic visualization of the ventricular chambers (cardiac ventriculography) has been reported in a previous publication by Lehman, Musser, and Lykens.[5]

Of 86 patients who have had satisfactory cardiac ventriculographic study specifically for the determination of the presence or absence of mitral valvular regurgitation, 32 have undergone surgical exploration of the mitral valve. The cardiac ventriculography studies of these 32 patients have been carefully graded with respect to the presence or absence of any regurgitant opacification of the left

atrium and the estimated quantitation of such opacification. The following method of grading is used: 0, no atrial opacification; 1+, trace; 2+, slight; 3+, moderate; and 4+, massive. These four grades of regurgitant opacification of the left atrium are illustrated in figure 1.

Any degree of regurgitant opacification of the left atrium was considered significant of mitral valvular insufficiency, since it has been our repeated observation in animal experiments and in a series of 110 cardiac ventriculographies (left) so far performed at our institution that the competent mitral valve will not permit of regurgitant opacification of the left atrium under the conditions of cardiac ventriculography as employed by us. In the grading of the patients presenting evidence of regurgitant opacification of the left atrium, due attention was paid to the size of the left atrial chamber, since, as Smith and co-workers [4] have pointed out, the degree of atrial opacification should be expected to vary with the volume of the left atrium, given the same degree of actual leak through the mitral valve. The grading of mitral insufficiency as adjudged by the surgeon at the time of exploration of the mitral valve is as follows: 0, no regurgitation; 1+, 1-5 cc. per beat; 2+, 6-10 cc. per beat; 3+, 11-40 cc. per beat; and 4+, over 40 cc. per beat.

While the ungloved examining finger has definite limitations in its ability to quantitate varying amounts of regurgitant stream issuing through the mitral valve, there is at present no better available method of surgical evaluation of dynamic mitral valvular leak. Any regurgitant jet through the mitral valve estimated at over 10 cc. per beat is considered at our institution to be of dynamic clinical significance. A leak of 1+ is represented by a very small but high-pressure jet issuing from the left ventricle with each systole; an insufficiency of 4+ is one in which a large surge of blood enters the atrium with each ventricular contraction.

Results

Of the 32 patients having both satisfactory ventriculographic study for demonstration of the presence and degree or absence of mitral regurgitation and for subsequent surgical evaluation of the status of the mitral valve, there were four patients who showed no atrial opacification, and at surgery these four were proved to have pure mitral stenosis without any mitral regurgitation. Of the remaining 28 patients, some degree of atrial regurgitation was noted on ventriculographic study in each, and, at surgical exploration of the mitral valve, each case presented some degree of mitral regurgitation.

Of these 28 patients presenting ventriculographic evidence of mitral insufficiency confirmed at surgery, there was exact correlation between ventriculographic estimation and surgical evaluation of the degree of mitral insufficiency in 17 patients. (In 2 patients, the grading was 1+; in 1, 2+; in 12, 3+; and in 2, 4+.) Of the remaining 11, the deviation between ventriculographic and surgical estimation was no greater than 1+ in 8. In one case, the ventriculographic estimation was two degrees greater than the surgical estimation, and in one case it was two degrees lower than the surgical estimation. In the other case, the operative report did not record a surgical grading.

Fig. 1.—Cardiac ventriculographies showing four grades of regurgitant opacification of left atrium. *A*, grade 1, *B*, grade 2, *C*, grade 3, and *D*, grade 4.

Comment

The determination of the degree of mitral insufficiency by cardiac ventriculography must obviously be an estimation only. Variations in the intrinsic

size of the left atrium and the dilution effects of the resultant wide range of blood volume in various left atrial chambers influence the opacification of the left atrium produced by regurgitation of radiopaque medium through the mitral valve. The procedure does not lend itself to exactitude of interpretation but rather to estimation only of the amount of regurgitant opaque substance issuing through the mitral valve. When it is considered that the surgical evaluation of the degree of mitral insufficiency also is an estimation, it is not surprising that some disparity exists between ventriculographic and surgical quantitation of mitral regurgitation. Variations in stroke volume, pulse rate, cardiac output, stability of blood pressure, and production of arrhythmias, which may be incident to either procedure, are factors which might well considerably modify for ventriculography on the one hand the degree of regurgitant opacification observed in the left atrium and on the other hand the strength, volume, and force of regurgitant stream of blood issuing through the mitral valve at surgery. It is, therefore, not astonishing that exact correlation between ventriculographic and surgical findings is not obtained in all cases.

Cardiac ventriculography appears to be a reasonably efficient procedure for the evaluation of reduction in mitral regurgitation following surgical procedures designed for the correction of mitral insufficiency. Figure 2 illustrates the preoperative ventriculographic study of a patient who, at surgery, presented mitral stenosis and a mitral insufficiency graded as 3+. The postoperative ventriculographic study, performed 14 days after combined mitral commissurotomy and cross polar plication of the mitral annulus, demonstrates in this case a marked reduction in the regurgitant opacification of the left atrium.

Summary

Cardiac ventriculography appears to provide a fairly accurate method for objective demonstration of the presence of mitral insufficiency. The reliability of this procedure in the quantitation of mitral insufficiency is apparently adequate for purposes of detection of clinically significant degrees of mitral insufficiency.

Considering operative findings at surgical exploration of the mitral valve as indicative of the presence or absence of mitral regurgitation, there were no false-positive ventriculographic diagnoses of mitral insufficiency in a study of 32 patients. In the same group of patients, there was exact ventriculographic correlation with surgical findings in 21 patients. While discrepancies between the estimated ventriculographic and estimated surgical quantitation of mitral insufficiency occurred in 11 cases, these discrepancies were not, in our opinion, of sufficient magnitude to invalidate cardiac ventriculography as a reasonably accurate clinical method of quantitating mitral insufficiency.

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Fig. 2.—Comparison of degree of regurgitant opacification in ventriculographic studies of left atrium before (*A* and *B*) and after (*C* and *D*) surgery for correction of mitral insufficiency.

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COMMISSUROTOMY FOR MITRAL STENOSIS

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The histories of 85 cardiac patients have been reviewed in order to define more sharply the indications and contraindications for mitral commissurotomy. The operation was completed in 72 patients without operative deaths, and three pregnant women in whom the operation was completed survived without fetal mortality. The effectiveness of the operation was clearly shown by comparing the preoperative and postoperative status of the patients with respect to the New York Heart Association functional classification. Case histories are given for five of these patients who died from 4 to 48 months after commissurotomy. The operation was not completed in 13 patients who presented various difficulties. Case histories were given for this group, in which four deaths occurred within the immediate postoperative period. In one instance the entire left atrium was found to have been converted into a smooth, bone-like mass through which entry was impossible. Mitral commissurotomy has become an effective, safe procedure for competent surgeons in well-selected patients. But when the heart is greatly enlarged and atrial fibrillation is present, when the auscultatory findings are not classic, when other valvular lesions exist (notably mitral insufficiency or aortic stenosis), or when the electrocardiogram shows left axis deviation, careful evaluation by other methods should be done, including fluoroscopy, phonocardiography, and catherization.

Commissurotomy is now an established procedure of merit in the treatment of mitral stenosis. General agreement has been reached regarding indications for surgery, except for the inclusion of asymptomatic patients. Contraindications are less well defined, and little attention has been directed toward those patients in whom surgical exploration of the mitral valve is done but in whom commissurotomy cannot be performed for various technical difficulties encountered at operation. The purpose of this report is to present the results of our experiences with mitral commissurotomy, to emphasize certain contraindications for operation, and to describe technical difficulties which preclude commissurotomy and contribute to mortality.

Population of Study

An analysis of the results of mitral commissurotomy in the treatment of patients with mitral stenosis has been made through a review and postoperative evaluation of patients operated on at Duke Hospital during the period from January, 1952, to July, 1956. Current follow-up data are available on nearly every patient. These patients constituted both private and public ward subjects at Duke Hospital, and the operations were largely done by one surgeon. Attention has also been directed to a group of patients who were operated on but in whom commissurotomy was not done. The cases of these latter patients demonstrate some of the problems encountered in mitral valve surgery.

During the period of 1952 to 1956, 85 patients were operated on at Duke Hospital, with mitral commissurotomy accomplished in 72. Exploration was done in 13 additional patients, but commissurotomy was not feasible because of an unsuitable valve or other technical difficulties. These 13 patients are considered separately. A second commissurotomy was performed in one patient 26 months after the initial operation because the valve had not been adequately opened during the first procedure.

The patients who underwent commissurotomy included 55 women and 17 men, a ratio of approximately three to one. On the basis of the New York Heart Association functional classification, preoperatively 41 patients were placed in class 2, 29 in class 3, and 2 in class 4. No patients in class 4 were denied operation. Three pregnant women were included in whom operations were performed at three, four, and five months of pregnancy without maternal or fetal mortality.

There were no operative deaths in the 72 patients. This reflects the opinion that patients in class 3, functional capacity, or in class 2 approaching class 3 are the best candidates for surgery.[1] Ellis and Harken [2] have reported a 0.6% operative mortality rate in their second 500 patients who were judged better risks for operation. Mitral commissurotomy has thus become a relatively safe procedure when done by competent surgeons, and the operative mortality is reaching a minimum in

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properly selected patients. Among the 72 patients in whom commissurotomy was done, there were 7 known deaths during the follow-up period. Among the 13 patients in whom only exploration was done, there were 4 operative deaths and 3 additional patients are known to be dead.

The average age at time of commissurotomy was 36 years, for both males and females. The age of the patients in class 3 and 4 averaged 38 years, as compared to 35 years for the patients in class 2. The youngest patient in the series was 19 years of age, while the oldest was 57.

Comparison of Preoperative and Postoperative Data

A history of rheumatic fever, considered to be reliable, was present in 31 (43%) of 72 patients. Seven of the 31 patients had chorea as the initial major manifestation of rheumatic fever; these were all women. A composite analysis of these patients reveals the average age at the first attack of rheumatic fever to be 11 years, with an asymptomatic period of 20 years, the onset of cardiac symptoms at 31 years, and commissurotomy performed 5 years later at 36 years of age. Mitral commissurotomy has become a means to interrupt the progression in the natural history of the disease, as documented by Bland and Jones.[3]

Analysis of some manifestations of rheumatic heart disease and mitral stenosis (table 1) revealed one or more episodes of congestive heart failure in 31 (44%) of the 72 patients, with this being more frequent in the patients in class 3 and 4 (27 [87%] of 31). Hemoptysis occurred in 27 (38%), being slightly more frequent in patients in class 3 than those in class 2. Auricular fibrillation occurred in 33 (46%) of the patients preoperatively. In nine patients having sinus rhythm, auricular fibrillation occurred during the immediate postoperative period. Conversion to normal sinus rhythm was accomplished in six of the nine patients. Systemic emboli, often multiple, occurred in 12 patients

TABLE 1.—*Preoperative Manifestations of Mitral Stenosis*

Class*	No. of Patients	Congestive Failure, No.	Hemoptysis, No.	Auricular Fibrillation, No.	Systemic Emboli, No.
2	41	5	15	16	4
3	29	25	12	16	8
4	2	2	0	1	0
Total	72	32	27	33	12

* New York Heart Association functional classification.

(16%) preoperatively; only 1 of these 12 patients has had an embolus postoperatively. In two patients without prior emboli, cerebral emboli occurred as a complication of operation. One patient without preoperative embolization died 41 months after operation from congestive heart failure and multiple systemic emboli.

A calcified mitral valve was found in 19 patients at operation (9 of 41 patients in class 2, 8 of 29 patients in class 3, and both patients in class 4). This was found twice as frequently in men as in women. Valvular calcification was detected roentgenographically in 21% of these patients preoperatively. Slight degrees of valve calcification had no appreciable effect on the subsequent course of the patient, while heavy calcification was noted in those patients who did less well.

TABLE 2.—*Preoperative Electrocardiographic Findings*

Class*	No. of Patients	Right Ventricular Hypertrophy	Right Axis Deviation	Digitalis Effect	Abnormal P Wave	Normal Finding
2	41	6	14	24	9	5
3	29	3	14	21	4	0
4	2	2	2	2	0	0
Total	72	11	30	47	13	5

* New York Heart Association functional classification.

Analysis of the preoperative electrocardiograms in the 72 patients (table 2) revealed right ventricular hypertrophy in 11 patients (15%), right axis deviation in 32 (42%), and abnormal P waves of the broad notched variety in 13 (18%). One patient presented the unusual finding of the Wolff-Parkinson-White syndrome, and another patient exhibited complete heart block. Five patients had normal electrocardiograms.

Radiologic examination in those patients who underwent commissurotomy nearly uniformly revealed an enlarged left atrium, prominent pulmonary vasculature, and, in 40%, right ventricular enlargement. No consistent change in heart size was observed postoperatively by comparison of roentgenograms. Postoperative electrocardiograms also showed little change from the preoperative tracings. Improvement in heart murmur, heart size, or electrocardiographic picture was less striking than symptomatic improvement in the successfully treated patient.[4]

In 33 patients, cardiac catheterization was performed preoperatively. In class 2, among 16 patients the average mean pulmonary capillary pressure was 21 mm. Hg (range 13-35 mm. Hg) with a rise to 31 mm. Hg after exercise (range 22-45 mm. Hg). The average mean pulmonary artery pressure rose from 37 mm. Hg (range 17-66 mm. Hg) to 54 mm. Hg (range 23-84 mm. Hg) with exercise. The 17 patients in class 3 and 4 had slightly higher pressures (average mean pulmonary capillary pressure of 24 mm. Hg with rise to 35 mm. Hg after exercise and average mean pulmonary artery pressure of 36 mm. Hg with rise to 59 mm. Hg). Most of the patients, on the basis of pressures, were in Holling and Venner's [5] stage 3 and 4 (valve size 0.5-1.0 sq. cm.).

Operative and postoperative complications included a depressive psychosis in 1 patient, auricular fibrillation in 9 patients, postcommissurotomy

syndrome in 19 patients, and cerebral embolism at time of operation in 2 patients (1 showed mild embolism without significant residua, while the other patient continues to have speech difficulty and mild motor disturbance two years postoperatively). The postcommissurotomy syndrome consisted chiefly of fever, pleuritis, and pericarditis in 19 patients (28%), and, as outlined by Dresdale and co-workers,[6] it might better be termed "postcardiotomy syndrome." Only 3 of these 19 patients had auricular muscle biopsy evidence of "active carditis." Eighty-three per cent of the patients in the series had "inactive" auricular muscle biopsies. The presence of positive biopsies had no obvious influence on the patients' subsequent clinical course. All seven patients who died after operation had negative auricular muscle biopsies.

Follow-up Data

Complete follow-up data to Jan. 1, 1957, are available for 70 of the 72 patients; the remaining 2 patients are known to be living but are not available for personal study. Postoperative observations have varied from 6 to 54 months, the mean duration of follow-up being 24 months for the patients in class 2 and 42 months for those in class 3 and 4. In table 3 the follow-up data are summarized. Ninety per cent of the preoperative class 2 patient group are improved, with 66% now placed in class 1. Of the preoperative class 3 and 4 patients, 74% are improved, 29% are grouped in class 1, and 39% are grouped in class 2. This degree of improvement is similar to that reported by Kuzman and associates,[7] Ellis and Harken,[4] and Bailey and Bolton.[8] The course of disease in two patients suggests that restenosis has occurred; one patient has been submitted to operation twice. Seven patients (two in class 2 and five in class 3 and 4 preoperatively) in the commissurotomy group are known to be dead.

Commissurotomy—Report of Cases

Case 1.—A 53-year-old woman (class 3) died approximately six months after operation in congestive heart failure with hyponatremia and, probably, pulmonary emboli. A calcified mitral valve and significant mitral regurgitation were found at operation.

Case 2.—A 22-year-old male (class 2) died 37 months postoperatively in acute pulmonary edema. This patient had combined aortic and mitral stenosis and auricular fibrillation; mitral regurgitation was noted at operation.

Case 3.—A 26-year-old woman (class 3), whose mitral stenosis was complicated by complete heart block, probably congenital, and mild diabetes mellitus, died about four months after operation in congestive heart failure. Mitral regurgitation was moderately increased after splitting the valve.

Case 4.—A 51-year-old woman (class 3) died approximately 48 months after operation of congestive heart failure. This patient presented a tight mitral stenosis, but, because of the funnel-shaped, calcified valve configuration, only partial relief of obstruction was possible. A history of cerebral embolus and auricular fibrillation was noted prior to operation, and her immediate postoperative course was complicated by a depressive psychosis severe enough to require temporary confinement in a mental hospital.

Case 5.—A 49-year-old male (class 3) with a heavily calcified valve noted at operation obtained temporary yet significant improvement in symptoms postoperatively. Death occurred 41 months after operation from severe congestive failure and multiple systemic emboli. Atrial fibrillation without history of embolism was present preoperatively.

Comment.—Five of these seven patients had atrial fibrillation, four had mitral insufficiency, four had calcified valves, two had very large hearts, and two had combined aortic stenosis and mitral valve disease. These patients presented most of the factors which have been considered to reduce the chance of operative success, such as marked cardiac enlargement, significant mitral insufficiency, calcified mitral valve, aortic stenosis, prolonged congestive heart failure, atrial fibrillation, and embolism.[9]

Table 3.—*Follow-up Data on Patients Who Received Commissurotomy for Mitral Stenosis*

Period Since Operation, Mo.	No. of Patients	Postoperative Functional Class				Improved, No.	Not Improved, No.	Deaths, No.
		1	2	3	4			
Preoperative Functional Class 2 (41 Patients)*								
6	4	1	2			2	1	1
12	8	7	1			8		
18	8	8	0			8		
24	4	1	2	1		3	1	
30	3	1	2			3		
36	7	3	3			6		1
42	4	3	1			4		
48	1	1				1		
54	2	2				2		
Total	41	27	11	1	0	37	2	2
Preoperative Functional Class 3 and 4 (31 Patients)								
6	3		1			1		2
12	2	1	1			2		
18	3	1	2			3		
24	3	2	1			3		
30	1		1			1		
36	2		1			1		1
42	4		3			3		1
48	9	2	2	3	1	6	2	1
54	4	3		1		3	1	
Total	31	9	12	4	1	23	3	5

* Current follow-up data not available for two patients.

Exploration Only—Report of Cases

The 13 patients in whom exploration was done but in whom commissurotomy could not be accomplished presented some interesting findings. The entire operative mortality has been contributed by this group. Less than 50% of these patients are alive, as compared to 90% of the patients who underwent commissurotomy. Of these 13 patients, 4 died in the immediate postoperative period and 3 died later, 1 at 1 month, 1 at 10 months, and another at 27 months after operation. Of the remaining six patients, five appear improved at the time of writing, not from surgery but through better patient cooperation and stricter medical management. Brief summaries of the cases of these patients follow.

CASE 1.—A 31-year-old male (class 3) was found at operation to have a thick, boggy, adherent pericardium, suggesting acute inflammation and edema. Operation was not attempted. This patient subsequently died three days after operation, and postmortem evidence showed acute rheumatic carditis, chronic congestive failure, and cirrhosis of the liver. His preoperative laboratory data included a normal hemoglobin level, white blood cell count, sedimentation rate, and unremarkable antistreptolysin-O titers (50-166 units). This illustrates the difficulty in diagnosing rheumatic carditis from laboratory data.

CASE 2.—A 45-year-old woman (class 3) was found at operation to have a valve so anatomically arranged that commissurotomy was not possible. There was only a minor degree of stenosis present. A large clot was noted in the atrium. The operation was further complicated by cardiac arrest for three minutes, during which time massage was done. The patient died five days postoperatively, and at autopsy a ball-valve thrombus was found obstructing the mitral orifice.

CASE 3.—A 27-year-old woman (class 2) at operation was found to have a calcified valve in which there was marked mitral insufficiency without significant stenosis. Her preoperative electrocardiogram had revealed right ventricular hypertrophy, and roentgenographically her heart was quite large. Both mitral systolic and diastolic murmurs were heard preoperatively. Postoperatively her course was complicated by hyponatremia, and death occurred seven days after operation from pulmonary embolism.

CASE 4.—A 30-year-old woman (class 4) at operation was found to have pronounced mitral insufficiency with a valve orifice estimated to be 3 cm. in diameter. The left atrium was found to be massively enlarged. Roentgenograms disclosed a very large heart, and the electrocardiogram revealed only atrial fibrillation and digitalis effect. Both mitral systolic and diastolic murmurs were present. She presented a history of cerebral embolization and for one month prior to surgery had been treated for suspected subacute bacterial endocarditis. Postoperatively her course was complicated by shock and oliguria and later by hyponatremia. She died 15 days after operation and, at postmortem examination, was found to have predominant mitral insufficiency without significant stenosis. The myocardium revealed healed Aschoff bodies. No evidence of subacute bacterial endocarditis was found at postmortem examination. Multiple small pulmonary emboli and one area of pulmonary infarction were demonstrated.

CASE 5.—A 38-year-old male (class 3) at operation had both mitral stenosis and insufficiency in a densely calcified valve whose configuration made opening hazardous in relation to possible increase of insufficiency. A stormy postoperative course was accompanied by atrial fibrillation, electrolyte disturbance, and a toxic reaction to quinidine. The patient died at home one month after surgery.

CASE 6.—A 35-year-old man (class 4) was found at operation to have the entire left atrium converted into a hard, smooth, calcified, bone-like mass which made entry impossible. This had been suspected preoperatively from the roentgenogram, and the patient was told that operation might not be possible. This patient died 10 months after operation in cardiac failure. At postmortem examination he was found to have severe mitral stenosis and a distended left atrium filled with calcified thrombus.

CASE 7.—A 50-year-old male (class 2) at operation was found to have such a heavily calcified valve that commissurotomy was not possible. Some mitral regurgitation was also present. Preoperative catheterization had revealed a mean pulmonary capillary pressure of 23 mm. Hg and mean pulmonary artery pressures of 39 mm. Hg at rest and 58 mm. Hg after exercise. This patient died 27 months after operation in severe congestive heart failure.

CASE 8.—A 47-year-old woman (class 2) had mitral stenosis and slight regurgitation at operation. Her valve was dilated by finger without any actual split being performed. Attempt to incise the commissure with a knife was not done for fear of causing considerable regurgitation due to the anatomic arrangement of the valve. On a careful medical program, this patient has done quite well for nearly six months.

CASE 9.—A 55-year-old woman (class 3) presented a loud mitral diastolic murmur with thrill, a mitral systolic murmur, atrial fibrillation, and a massively enlarged heart. Cardiac catheterization revealed a mean pulmonary capillary pressure of 16 mm. Hg and a rise of mean pulmonary pressure from 20 to 29 mm. Hg with exercise. At operation her mitral valve area was at least 3 sq. cm. in area without significant mitral regurgitation. In the year since operation her status has improved slightly with a strict medical regimen.

CASE 10.—A 46-year-old woman (class 3) preoperatively presented murmurs of mitral stenosis and insufficiency, atrial fibrillation, enlarged left atrium and right ventricle by fluoroscopy, and right axis deviation by electrocardiogram. Cardiac catheterization revealed a mean pulmonary capillary pressure of 19 mm. Hg and mean pulmonary artery pressures of 25 mm. Hg at rest and 36 mm. Hg after exercise. Operation disclosed predominant mitral insufficiency without significant stenosis. She remains in class 3.

CASE 11.—A 42-year-old male (class 2), who presented atrial fibrillation and a history of cerebral embolus, was found at operation to have primarily mitral insufficiency. Both mitral systolic and diastolic murmurs were present. The auricular appendage was removed. Postoperatively, no further emboli have been noted. Attempts to restore normal rhythm have failed because of serious manifestations of quinidine toxicity. The patient remains unimproved.

CASE 12.—A 43-year-old woman (class 3), who preoperatively had mitral systolic and diastolic murmurs, moderate left atrial enlargement, and a normal electrocardiogram, was found to have moderate mitral insufficiency with no significant stenosis. Except for the postcardiotomy syndrome, she has done well postoperatively.

CASE 13.—A 37-year-old man (class 2), who had a history of cerebral embolus, was found at surgery to have predominant mitral insufficiency with slight mitral stenosis. The auricular appendage was removed. The patient has had no further emboli and is working 36 months postoperatively. He also has auscultatory evidence of probable aortic insufficiency.

Comment.—Of the 13 patients whose hearts were explored without commissurotomy, there were 6 who had primarily mitral insufficiency without significant stenosis, one who had neither significant stenosis nor insufficiency, 3 whose valves were not amenable to correction, 1 with a too heavily calcified valve for commissurotomy, 1 with a densely calcified atrium precluding surgical entrance, and, finally, 1 with acute rheumatic pancarditis. Although Ferrer and associates [10] have discussed the clinical differentiation of mechanical block at the mitral valve from myocardial insufficiency by the catheter technique, the practical problems presented by this group of 13 patients have generally received little attention in clinical reports concerning the results of commissurotomy.

Need for Surgery

In retrospect, some of these patients should not have been operated on, particularly the young man with active rheumatic carditis or the man with the tremendously calcified atrium which made operation hazardous, although the latter patient's

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symptomatology was so severe that exploration at least was considered worthwhile. In relation to the problem of mitral insufficiency, catheterization of the left side of the heart now provides a tool for evaluating patients suspected of having this lesion.[11] Unusually shaped mitral valves cannot always be detected, although the presence of massive calicification should make this suspected. The last case in the group above was a variant of those discussed by Harvey and associates [12] in relation to the problem of myocardial insufficiency. As outlined by Harvey and associates, these cases can be detected by physiological studies. Consequently, cardiac catheterization of both right and left sides should be resorted to more frequently for objective evaluation especially in the problem patient. The phonocardiogram has been studied in relation to the correlation of Q-1 minus 2-O.S. times with the degree of mitral block and can be helpful for the screening of patients for mitral commissurotomy.[13] (The Q-1 interval refers to the time from the onset of the QRS wave to the onset of maximal vibration of the first heart sound. The 2-O.S. interval is the time from the onset of the second sound to the opening snap. In our experience, a 2-O.S. time less than 0.09 seconds and a Q-1 minus 2-O.S. value greater than minus one, present in the same patient, are suggestive of significant mitral stenosis.)

Black and Harken [14] have enumerated some helpful clues in the determination of the need for surgery in patients with mitral stenosis. Episodes of failure, roentgenographic evidence of pulmonary hypertension, electrocardiographic evidence of right ventricular hypertrophy, difficulty with coitus, episodes of systemic emboli, and severe symptoms in pregnancy are all factors which, as in our experience, have been helpful in the selection of patients for operation. A thorough and carefully done history and physical examination, supplemented by routine diagnostic aids, are, as emphasized earlier by Kuzman and his associates,[7] the basic tools in the evaluation of candidates for mitral surgery.

Little difficulty is offered by the symptomatic patient who presents the classic findings of mitral stenosis with accentuated first heart sound, opening snap, rumbling mitral diastolic murmur and thrill, loud split pulmonary second sound, right axis deviation by electrocardiogram, enlarged left atrium, prominent pulmonary artery, and hypertrophy of the right ventricle. When the heart is greatly enlarged and atrial fibrillation is present, when the auscultatory findings are not classic, when other valvular lesions exist (notably, mitral insufficiency or aortic stenosis), or when the electrocardiogram shows left axis deviation, careful evaluation by other methods, such as with phonocardiogram and catheterization of the right and left sides of the heart, should be done before subjecting the patient to surgery. Fluoroscopic examination may sometimes furnish valuable clues.

The demonstrated safety and effectiveness of mitral commissurotomy in well-selected patients who have mitral stenosis makes accurate evaluation of all suspected cases obligatory in order to choose the suitable candidate and to avoid ill-advised surgery with its higher mortality rates. Surgical exploration of questionable cases should be avoided through first utilizing every diagnostic aid available, and only then, in a minimum number, will exploration be justified.

Summary

Mitral commissurotomy was performed in 72 patients at Duke Hospital with no operative deaths. Exploration was done in 13 additional patients, but commissurotomy was not feasible because of an unsuitable valve or other technical difficulties. Forty-one patients were placed in functional class 2 and 31 in class 3 and 4. Three pregnant women were operated on without maternal or fetal mortality. A prior history of rheumatic fever was present in 43% of the patients, the average age at onset of the first attack being 11 years followed by an asymptomatic period of 20 years. Symptoms of rheumatic heart disease appeared at an average age of 31 years, and commissurotomy was performed at an average age of 36 years. One or more episodes of congestive heart failure, hemoptysis, and atrial fibrillation occurred in nearly half of the patients preoperatively.

Follow-up in 70 of 72 patients revealed impressive improvement in symptoms; 90% of the patients grouped preoperatively in class 2 improved, and 66% were placed in class 1. Seventy-four per cent of the patients in class 3 and 4 were improved, with 29% placed in class 1 and 39% in class 2. Improvement in heart murmur, heart size, or electrocardiographic tracing was less impressive than subjective improvement in successfully treated patients. Operative complications included the postcardiotomy syndrome, which occurred in 26% of the patients without correlation to the auricular muscle biopsy evidence of rheumatic activity. Seven patients are known to be dead, and these patients had clinical findings suggesting less chance of operative success, namely, the association of mitral insufficiency, aortic stenosis, very large hearts, valve calcification, and atrial fibrillation.

The cases of 13 patients whose hearts were surgically explored without commissurotomy reveal some important problems in terms of selection and technical difficulties at operation. This group constituted the entire operative mortality (four deaths) and a low survival rate (less than 50%).

Mitral commissurotomy has become a safe and effective procedure in well-selected patients and offers a means of interrupting the progressive symptoms of rheumatic heart disease and mitral stenosis.

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EFFECT OF SAFFLOWER OIL EMULSION ON SERUM CHOLESTEROL LEVELS IN YOUNG ADULT MALES

ITS USE AS A SUPPLEMENT TO A NORMAL DIET

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The effect of safflower oil emulsion given as a supplement to the usual diet was studied in 24 young men, weekly samples of blood being taken and analyzed for total cholesterol. After an initial period of five weeks, the subjects were divided into two groups who received either safflower oil emulsion or a placebo for seven weeks. At the end of that time the two groups were reversed, so that those who had received the placebo now received emulsion and vice versa. Neither the subjects nor the supervising physicians knew which preparation a given subject was receiving at a given time. The emulsion, containing 65% safflower oil, was given in 15-cc. doses five times daily to a total of 75 cc. each day. Used as a supplement to the usual diet under these conditions, it had no demonstrable depressant effect on the total serum cholesterol levels.

Vegetable oils have received attention recently because of their effect of lowering the levels of serum lipids in man. It seems definitely established that the careful substitution of unsaturated vegetable oils for saturated animal fats in human diets will produce a sustained reduction in total serum cholesterol level.[1] Several unsaturated vegetable oil emulsions are now available. The temptation on all sides has been to assume that they would effectively reduce the serum cholesterol level, when given as dietary supplements, without much change in the patient's regular diet.

The work of several investigators [2] after the first data from Kinsell and Michaels and Ahrens and others [3] has indicated that the amount of fall in the serum cholesterol level is closely allied to the degree of unsaturation of the particular vegetable oil supplied to the subjects under investigation. This has caused a focusing of attention on safflower oil because of its high percentage of linoleic acid and high degree of unsaturation. With an iodine number of about 143, safflower oil is the most unsatu-

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rated vegetable oil available today. But, despite the widespread manufacture and prescription of safflower oil emulsions and despite the fact that large numbers of patients are taking these supplements to their normal diets in hopes of reducing their serum cholesterol levels, very little experimental evaluation of their value as such has to date been published.

Ahrens and others [1b] has pointed out the close parallel between the levels of total cholesterol, free cholesterol, and phospholipids in the serum and have shown that a change in total serum cholesterol level is a satisfactory index of a change in levels of all three factors.

The purpose of the present study, therefore, was to investigate the effect on the total serum cholesterol level in 24 normal young male subjects of a safflower oil emulsion given as a supplement to the usual diet.

Material and Method

Selection of Subjects.—Twenty-four young male medical students, averaging 24 years of age (range 22 to 26), were selected to serve as paid volunteers. None had a history or clinical evidence of atherosclerosis or of disordered thyroid function.

Control of Diet.—The habitual diet of each subject was determined by questionnaire before the beginning of the study. The following average composition of the diet was established: total calories, 2,350 (range 1,900 to 3,100); calories derived from fat, 42% (range 34% to 47%); and total daily fat intake, 109 Gm. (range 88 to 119), the great majority of which was animal and hydrogenated vegetable fat. All the subjects were required to adhere to their usual diets as closely as possible during the control and experimental periods. Use of vitamin supplements, the only medication taken previously by any of the subjects, was discontinued.

Experimental Design and Method.—During the entire experiment, serum was obtained weekly by venipuncture from each subject during the fasting state. Each sample was analyzed for total cholesterol level by a modification of the method of Bloor. In general, the cholesterol level determinations were performed on fresh serum on the same day. Occasionally, the serum was frozen at −15 C, and the determination was performed one to six days later.

After a preliminary control period of five weeks, the subjects were divided into two groups. They were matched according to nearest average serum cholesterol level seen thus far in the experiment and were placed alternately into one or the other group. One group of 12 subjects then received a 65% safflower oil emulsion, 15 cc. five times daily (total 75 cc. a day). Thus, these subjects ingested approximately 48 Gm. of unsaturated vegetable oil daily, creating a ratio of unsaturated to saturated fat in their diets of about 1:2. This daily dosage of 75 cc. was chosen because it is the usual maximum dosage advised to produce the desired depressent effect on the serum cholesterol level.

The other group of 12 subjects were given, in the same dosage, a placebo, resembling the safflower oil emulsion in taste and consistency. The compositions of the two materials are as follows: The safflower oil emulsion contained 65% safflower oil, 8% sucrose, and 22% water; as emulsifying agents, polyethylene glycol distearate, polysorbate 80, sorbitan monooleate, sodium lauryl sulfate, and polyoxyethylene sorbitan monopalmitate; artificial flavor; as preservatives, 0.1% sodium benzoate and 0.05% benzoic acid; and as antioxidants, 0.0065% propyl gallate and 0.003% citric acid. The 75-cc. dosage supplied 450 calories. The placebo contained, per 1,000 cc., 100 Gm. of granulated acacia, 1 Gm. of sodium benzoate, 1 cc. of vanilla tincture, 800 Gm. of sucrose, and distilled water. The 75-cc. dosage supplied 240 calories. Standard 15 cc. tablespoons were dispensed to all. The timing of the doses, whether before, during, or after meals, was not regulated.

The experiment was conducted by the double-blind method. Neither the subjects nor the supervising physicians were told whether each individual subject was taking the safflower oil emulsion or the placebo.

After seven weeks of therapy, the two experimental groups were reversed. The group that had taken the safflower oil emulsion then took the placebo, and those who had taken the placebo then took the safflower oil emulsion.

Results

During all phases of the experiment, there was a marked variability from week to week in individual serum cholesterol levels. The average cholesterol level of all 24 subjects, however, remained fairly stable from week to week during the control period. The over-all average total serum cholesterol level in these subjects over a period of five weeks, without therapy, was 192 mg. per 100 cc. (range 117 to 284).

When, after the control period, 75 cc. of the safflower oil emulsion was added daily to the regular diets of 12 subjects, over a period of seven weeks, there was no decrease in the average total serum cholesterol level (table 1). In fact, in 11 of 12 subjects, the average cholesterol level while the safflower oil emulsion was taken was higher than it had been during the control period. The average increase was 17 mg. per 100 cc. (range 1 to 36).

However, the group of 12 subjects who went from a control period to a period of seven weeks during which the sucrose placebo was added to their diets (table 2) also showed a rise in average serum cholesterol level; in 11 of 12 subjects this rise was 23 mg. per 100 cc. (range 6 to 44).

When, at the end of seven weeks, 12 subjects were switched from safflower oil emulsion to placebo, the gradual trend upward in serum cholesterol level continued (table 1). Similarly, in the other 12, when the safflower oil emulsion was substituted for the placebo, the average total serum cholesterol level continued to rise slowly (table 2).

There was no depressant effect on the total serum cholesterol level to be observed from the addition of 75 cc. daily of the safflower oil emulsion to the regular diets of these subjects (see figure).

The safflower oil emulsion used was well tolerated by the subjects. One subject out of each group of 12 developed slight nausea and loose stools during the first week of therapy, but these symptoms did not persist. The subjects reported a slight weight gain (average 2 kg. or 4.5 lb.) during the period of addition of the emulsion to their regular diets.

Comment

The ability of supposedly normal young male subjects to respond by a lowering in the serum cholesterol level to a substitution of unsaturated vegetable oil for saturated fat in the diet has been previously established by several other investigators.[2a] The similar effectiveness of safflower oil as a practical supplement to a normal diet, however, has not yet been satisfactorily demonstrated. Armstrong and co-workers[4] have recently published the results of an experiment in which safflower oil, in about the same daily quantity used here, was added as a supplement to the diets of medical students. These authors reported a significant lowering of the serum cholesterol level, but their data were the result of only a nine days' study, with but two determinations of cholesterol level, one before and one after.

The present study failed to show any depressant effect on the serum cholesterol level. There was, in this study, a considerable variability in the individuals' cholesterol levels from week to week. Such variability has appeared to us to be less in older subjects and in patients with elevated serum cholesterol levels and points up the necessity, often neglected, of establishing an adequate control or base-line series of determinations in any such study. A control series of at least five weekly determinations, as done here, would seem necessary.

The reason is uncertain, in the present study, for the gradual rise in the average serum cholesterol level over an 18-week period in both of the groups (control–safflower oil–placebo group and control–placebo–safflower oil group). The increase in calories consumed and seasonal influence (the

TABLE 1.—*Weekly Serum Cholesterol Level Determinations in Milligrams per 100 cc. Over Three Treatment Periods in Twelve Subjects (Group 1)*

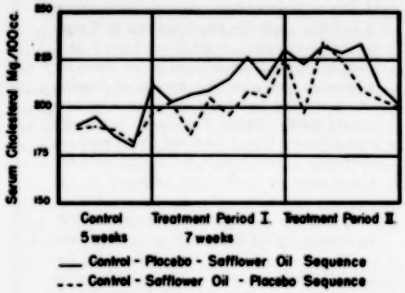
	Control						Safflower Oil Emulsion							Placebo					
Case / Week No.	1	2	3	4	5	Av.	6	7	8	9	10	11	12	Av.	13	14	15	16	17	18	Av.
1	243	248	256	276	263	257	228	260	248	246	307	248	326	266	256	311	267	303	288	304	282
2	228	243	222	204	210	221	228	222	252	235	...	276	232	241	164	273	267	226	243	229	234
3	198	222	191	228	210	210	198	204	196	216	173	210	243	206	222	260	239	226	268	229	231
4	216	194	198	210	204	204	222	194	222	204	256	171	225	215	219	232	247	175	198	229	218
5	198	184	228	176	235	204	235	189	235	222	204	260	256	229	198	198	214	273	229	217	223
6	...	187	184	162	222	189	216	189	189	196	189	207	213	200	213	229	229	202	183	159	203
7	204	169	184	179	201	187	204	204	216	225	239	256	204	221	187	...	229	186	223	214	206
8	164	198	187	146	189	177	204	171	201	179	171	179	204	187	187	202	229	198	183	177	195
9	159	176	179	184	184	176	174	154	198	162	184	159	216	178	187	173	188	198	183	198	188
10	150	173	146	159	159	157	194	179	179	192	210	189	210	193	194	288	223	214	183	193	216
11	173	159	157	146	150	157	189	155	204	164	184	179	204	183	189	247	202	183	154	152	188
12	143	121	129	117	134	129	146	113	117	93	146	143	155	130	143	175	166	123	168	125	150
Over-all av.	189	190	188	182	197	189	203	186	205	196	208	206	226	204	197	234	224	209	204	200	211

TABLE 2.—*Weekly Serum Cholesterol Level Determinations in Milligrams per 100 cc. Over Three Treatment Periods in Twelve Subjects (Group 2)*

	Control						Placebo							Safflower Oil Emulsion							
Case No. / Week No.	1	2	3	4	5	Av.	6	7	8	9	10	11	12	Av.	13	14	15	16	17	18	Av.
1	216	284	235	239	276	250	301	243	276	266	252	303	268	256	316	267	311	287	252	223	278
2	256	222	222	194	280	235	303	306	252	280	309	228	289	304	284	300	239	226	288	229	241
3	235	228	222	198	246	226	300	243	284	309	257	280	266	270	235	273	309	273	236	232	252
4	194	194	...	194	222	201	216	225	304	228	216	225	232	222	219	250	214	305	220	198	218
5	196	207	169	194	216	190	210	186	210	194	210	216	228	209	226	239	232	229	252	223	233
6	184	194	157	184	198	183	210	198	192	179	235	210	235	208	210	232	220	208	173	202	208
7	155	159	189	189	216	182	216	198	171	173	216	228	187	198	189	202	208	220	247	175	207
8	173	194	169	204	164	181	164	179	179	189	192	204	216	189	216	220	214	247	214	177	215
9	179	184	184	196	143	173	198	204	204	235	232	216	210	214	225	273	229	297	214	202	240
10	167	171	173	153	194	172	194	184	210	207	206	189	204	207	201	198	229	202	175	214	205
11	179	155	145	173	184	167	150	173	173	173	210	184	194	180	194	183	208	188	198	220	199
12	155	150	173	143	179	160	148	150	150	162	159	125	179	153	164	183	177	202	147	133	168
Over-all av.	191	195	185	180	212	194	208	207	209	215	226	215	220	214	223	232	228	233	211	202	223

experiment lasted from mid-summer to early winter) may have played a part. The design of the experiment, however, with each subject serving as his own control, compensated in part for this.

At any rate, the use of safflower oil in the maximum dosage usually recommended for therapeutic purposes produced no depressant effect on the levels of serum cholesterol in these normal young male subjects maintained on their usual diets.



Mean total serum cholesterol levels in two groups of 12 subjects each.

Our results are not to be construed as denying the possible effectiveness of safflower oil as a supplement to diets which have been markedly reduced in their content of animal fat.

Further studies on dietary supplements of safflower oil, other unsaturated vegetable oil preparations, and combinations of these with substances supposed to reduce the serum cholesterol, in normal young subjects, in older subjects with arteriosclerosis, and in subjects with elevated serum cholesterol, are under way and will be reported on later.

Summary

Twenty-four young male subjects were studied during a three-phase experiment lasting 18 weeks. After a five-week control period, they were given, in sequence for six-week to seven-week periods, a placebo, and a daily dietary supplement of 75 cc. of a 65% safflower oil emulsion. The safflower oil emulsion as a supplement to the usual diet had no demonstrable depressant effect on the total serum cholesterol levels of these subjects.

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BOTULISM.—[This is] a disease which, owing to its spectacular nature and high case-fatality rate, appears to occupy a place out of proportion to its frequency as a cause of death in some regions. Without exception, botulism is caused by carelessness in the preparation and preservation of vegetable and animal foods. Local customs of eating such food uncooked, in the form of salads, watery conserves, poorly cured or inadequately smoked pork and salted fish products create the botulism problem. The risk of botulism exists wherever the telluric incidence of Type A, B, and E spores is high. . . . In California, with an epidemiological intelligence service attuned to botulism, the casual toxin or type of anaerobe was proved in only 69 (33.9%) of 203 outbreaks. . . . All recent observations support as more plausible the explanation that dissimilarities in the foodstuffs customarily responsible for botulism in various parts of the world (processed meats—especially ham, sausages, and other pork products—in Europe; home-preserved vegetables and fruit in the USA; fish in the USSR) may be major determinants of regional case-fatality rates.—K. F. Meyer, M.D., The Status of Botulism as a World Health Problem, *Bulletin of the World Health Organization*, vol. 15, no. 1-2, 1956.

BLOOD BANKING FOR INTRACARDIAC SURGERY

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The amount of heparinized blood needed during an intracardiac operation ranges from 6 half-liter units for small patients to 9 units for large patients; in addition, from 1 to 4 units of citrated blood must be on hand for supportive use after the period of heparin administration has been terminated by protamine administration. Experience has shown that ordinary blood from the blood banks is satisfactory for citration, but the heparinized blood must be fresh. It is collected on the morning of the operation by running 470 ml. of blood into a silicone-coated vacuum bottle containing 1,500 U. S. P. units of heparin in 30 ml. of saline solution. Before the bypass is instituted, the patient also receives by direct intravenous injection 1,500 U. S. P. units (approximately 15 mg.) of heparin per 500 ml. of blood volume. Throughout the operation there is continual calculation of blood loss and replacement. Experience has made it possible gradually to eliminate many tests, such as plasma hemoglobin determinations, that formerly were made repeatedly during the period of heparinization. The large amounts of blood required make it necessary to plan months ahead, and the crossmatching for a given patient is done two or three days before the expected date of operation. This program should be the primary concern of experienced, trained clinical pathologists and can develop a spirit of cooperation in all concerned, including the professional donors.

Intracardiac surgery with use of a heart-lung bypass machine makes special demands in respect to provision and management of blood. To meet these requirements in the use of a Gibbon-type pump-oxygenator, members of the Mayo Clinic Blood Bank and the appropriate surgical team employ the procedures to be described here.

Knowledge of two statistical statements is needed for proper evaluation of the problem: Some 15,000 half-liter units of whole blood are used annually at the clinic; and of this amount, approximately 3,000 units (20%) are used by the bypass machine alone. Since it is necessary that this blood be fresh, heparinized, and, of course, of the proper group and type, the problem of procurement obviously is a major one.

Fortunately the clinic has a background of some 43 years of experience in providing blood for transfusions. Over this period a list of a considerable number of professional donors has been built up. The description of the details of this procurement is beyond the scope of this paper, since they include such broad psychological concerns as the development of an *esprit de corps* among people not acquainted with one another. For example, as our immediate community is relatively small, we have some donors who drive from 200 miles away to donate blood.

Perhaps our greatest problem is the disparity between the blood types available and the types needed. Our donor group contains a large percentage of Scandinavians, who are predominantly of type A; but our patients, many of whom come from greater distances, are chiefly of type O.

Scheduling of Patients

In order to have a basis for arrangements with donors, a fairly rigid schedule of operations for a three-month period is submitted to the blood bank. Included are each patient's blood group and type and his age, surface area, and sex. This list is compared with the lists of available professional donors who will be eligible for call on the tentative date of operation. If enough suitable donors are not available on such a date, another date is suggested to the surgeons by the clinical pathologists who operate the blood bank. This final date, then, is considered definite—although, of course, it may be altered at the last minute owing to changes in the patient's clinical course, transportation, or will. In such event a substitution is made, if feasible.

Copies of the accepted schedules and substitutions are distributed widely throughout the clinic: Within the laboratory system alone they go to the bulletin boards of the clinical pathology laboratory, to the clinic blood bank, to the hospital blood bank, to the typing and grouping laboratory of the clinic, and to the hospital laboratories.

Since the tentative three-month schedule frequently is based on groupings done elsewhere that sometimes are at variance with our own, the pa-

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tient's blood is grouped and typed here a few days before the date set for operation. The grouping is performed as a slide test and read with the unaided eye. Both cells and serum are examined, and the grouping is determined independently by two workers. The Rh determination also is made as a slide test, and the blood is considered Rh-positive if its cells react against Rh_0 antiserum. When results are doubtful, tube tests are performed.

Calculation of Blood Needs

Necessity for Heparinization.—A major problem is that the machine requires blood collected in a minimum of anticoagulant solution. The usual 120 ml. of A. C. D. solution per unit of blood is too much; the amount of citrate in the solution may produce fatal hypocalcemia. Therefore, blood must be collected in heparin solution, of which 30 ml. of 0.9% sodium chloride solution containing 1,500 U. S. P. units of heparin is used per unit of blood. The bottles are siliconized. One period during the surgical procedure, designated as the "heparinization period," includes the time the patient is undergoing bypass and also the time required for examination, connection of cannulas, observation of results, and so on, extending to the time of protamine administration. During this period only heparinized blood is administered. Because heparinized blood must be fresh when used, the actual amount needed for each patient must be calculated fairly closely.

Separate Factors.—The quantity is derived from three sources: the amount of blood needed for the machine and flow lines; the amount of blood needed for transfusion from the machine during bypass; and the amount of blood needed by the anesthesiologist for transfusion during those brief parts of the period of heparinization before and after bypass.

1. For Machine and Cannulas: The oxygenation system of the Gibbon-type machine is a series of screens, of which the number used is varied in proportion to the calculated oxygen needs of the patient. Nomograms based on the patient's height, weight, and surface area characteristics indicate the blood flow required, which determines the number of oxygenator screens needed; and from that the amount of blood necessary to cover the screens and to fill the flow lines and reservoirs is figured. As an example, for a tiny patient with a surface area of 0.2 to 0.24 sq. m., three screens are needed; therefore, the machine and flow lines require 2 liters of blood.

2. For Transfusion from Machine During Bypass: For the same patient, an extra 500 ml. of blood is kept in the reservoirs of the machine for transfusions from the machine during bypass. For any patient whose surface exceeds 0.24 sq. m., 2 units of blood are kept in the wells.

3. For Transfusion During Heparinization Period but Off Bypass: One unit of heparinized blood is reserved for transfusion before and after bypass in all cases.

Totals and General Considerations.—In sum then, for a patient of 0.2 to 0.24 sq. m. surface area, a total of 6 units of heparinized blood is required. At the other extreme, a patient of 2 sq. m. or more requires 14 oxygenator screens for proper oxygenation, and thus 6 units of blood for the machine and cannulas, another 2 for transfusion from the machine, and 1 for off-bypass transfusion as mentioned, making a total requirement of 9 units of heparinized blood. With increasing experience, the needs for heparinized blood have constantly decreased, but this range of 6 to 9 units is considered to be the minimum at the moment.

However, additional blood must be on hand for supportive use after the heparinization period. Citrated blood is used for this purpose. For the tiny patient of 0.2 to 0.24 sq. m. surface area, 1 unit is reserved for this purpose; for the other extreme of 2 sq. m. of surface area, 4 units are kept on hand. The belief governing the use of citrated blood has undergone a change with time. Early in the course of experience with open-heart operations at the clinic, it was thought necessary to utilize freshly collected citrated blood. Gradually, however, slightly older blood was used without mishap—at first an occasional unit, then more and more, so that 5-day-old and then 10-day-old citrated blood was found to be satisfactory. Now ordinary bank blood has been found entirely acceptable for this purpose.

Crossmatching

Two or three days prior to the expected date of operation, 20 ml. of blood is withdrawn from the patient for crossmatching. An appropriate number of donors are given appointments to present themselves at the blood bank on the day preceding operation. We have considered it wise to schedule an extra donor for each case, because one might fail to appear. (After the need for heparinized blood has been met, blood from any additional donors is collected in A. C. D. solution.)

From each donor 5 ml. of blood is withdrawn for crossmatching with that of the patient, for a Venereal Disease Research Laboratory (VDRL) slide test, and for hemoglobin determination. Major and minor crossmatches are then carried out between blood of the patient and of each donor at three temperatures: 11, 25, and 37 C. Likewise, autoagglutinin tests are performed on all bloods at these temperatures. No crossmatching between donors has been done, and in more than 400 operations performed with utilization of the Gibbon-type pump-oxygenator no problems have arisen from interdonor incompatibility.

If, prior to operation, the blood of any donor is found incompatible, a substitution is made from a list compiled for this purpose and the donor is informed that he need not appear. This situation has arisen twice.

Blood Collection

The donors return for phlebotomy between 7:00 and 7:30 a. m. on the day of operation. This is carried out in the usual way: from each donor 470 ml. of blood is drawn through a 17-gauge disposable needle and disposable polyethylene tubing into a silicone-coated vacuum bottle containing 1,500 U. S. P. units of heparin in 30 ml. of saline solution.

The unit of blood, which may be citrated rather than heparinized, is collected in 120 ml. of A. C. D. solution (U. S. P. and N. I. H. formula B).

During the collection a special messenger is available to carry all the blood directly to the operating room wherein the operation is to be performed. This requires 10 to 15 minutes. On arrival there, the bottles of heparinized blood are placed in a water bath at 37 C to prevent cooling until they are ready to be used by the machine. Of course the blood should never be heated to a higher temperature. (Citrated blood never is placed in the water bath.) As soon as the machine has been completely assembled and the thoracotomy performed, the heparinized blood is emptied gently into the screen-filtered receptacle of the oxygenator machine.

Management of Blood Characteristics and Balance in Patient During Operation

Prevention of Osmotic Disorders.—The 30 ml. of saline solution included with each unit of heparinized blood filling the machine and otherwise in readiness—a total of 180 to 270 ml. of saline solution in the 6 to 9 units required—is a minimum irreducible at this time. A real danger would be present should this fluid leave the vascular system. For example, the resulting relatively small change in osmotic pressure may produce a fatal degree of pulmonary edema. In order to prevent this, concentrated (25%) salt-poor human serum albumin (generously supplied by the American Red Cross) is poured along with the blood into the machine's receptacle at the rate of 1 ml. of albumin to each 4 ml. of saline solution.

Recovery from Unused Tubing.—The machine utilizes teflon tubing of 0.5-in. caliber for all the major flow lines, and a tube of smaller bore is used in the coronary sinus suction system. Since, therefore, the cannulas themselves hold a considerable quantity of blood, the contents of any excess tubing removed in the process of attaching the machine's vessels to those of the patient are carefully emptied into the machine's reservoir.

Injection of Heparin.—Theoretically, prior to institution of bypass the patient should be given intravenously enough heparin to match that used by the machine—that is, 1,500 U. S. P. units (approximately 15 mg.) per 500 cc. of blood volume. In actual practice the dosage is 3 mg. per kilogram of body weight. Half of this is given intravenously three minutes before the arterial cannulas are inserted and the other half when the two vena caval cannulas are inserted.

Maintenance of Balance.—Throughout the operation there is continual calculation of blood loss and replacement; in fact, a blood balance sheet is kept for this accounting. The blood lost to suction and that removed for tests are measured directly. All gauze and toweling are weighed, dry and wet, and their blood content calculated. The blood in the operative field is estimated. From these data is figured the net blood loss, which is tallied against net blood supplied; and thus the balance is determined. In order to waste as little as possible of the heparinized blood on hand, this balance is made up during the period of heparinization by transfusion from the machine. When feasible, the patient is weighed before and after operation as accurately as possible to check on blood loss. These weights are tabulated also. When the patient's blood balance has been evened at the end of perfusion, the cannulas are removed and 3 mg. of protamine per kilogram of body weight is injected intravenously.

Blood for examination of plasma hemoglobin is drawn from the machine after disconnection in order to ascertain whether trauma to blood has been excessive. Results of these determinations have always been less than 40 mg. per 100 ml., the lower limits of accurate determination by the method of testing. (The method used is simple, rapid, and direct, and although rough it has been found to yield consistent results: A mixture of 2 ml. of plasma and 8 ml. of isotonic sodium chloride solution is read directly in a photelometer, with use of distilled water as a control. The reading is then compared with a table based on chemical determinations. Readings of less than 40 mg. per 100 ml. have been found in normal individuals.) The operative wound is then closed and the patient is returned to a special postoperative recovery unit in the hospital. The heparinized blood remaining in the machine is discarded.

Tests Made.—During the early experience with open-heart surgery with use of extracorporeal circulation, many more laboratory tests were made than are carried out at present. These discontinued tests and the number of times they were originally performed during perfusion are as follows: plasma hemoglobin determination, seven; cell volume determination (hematocrit), six; bilirubin (both direct and indirect), three; sodium, potassium, chloride, and carbon dioxide, four; urea and creatinine, four; total proteins, with albumin and globulin dif-

ferential, five; hemoglobin, red blood cell, white blood cell, and platelet counts, four. Clotting time was determined with great frequency. Obviously these tests were carried out at the beginning because so much of what was happening to various blood factors during perfusion was unknown. As experience has been gained, tests have been discontinued one by one, until now only the examination of plasma hemoglobin at the end of perfusion remains a routine procedure, as a check on hemolysis within the system.

Comment

We have attempted to eliminate every frill of testing and every type of unnecessary test so that we can concentrate on the essential tests in order to provide proper blood for the patient. Perhaps the key to the whole process is the fact that the procurement of blood for these and all other operations is the primary concern of experienced, trained clinical pathologists and is not left to technicians and nonprofessional personnel or those who procure blood as a sideline.

Although we do not wish to imply that all of the problems concerned with this intricate and involved procedure have been solved, we do feel that we have, for the moment at least, a practical solution to the problems presented to our blood bank. It is of interest to note in closing that in the experience with over 400 operations with the pump-oxygenator performed as of this writing, not a single donor has failed to keep his early-morning appointment at the blood bank.

RESISTANCE AND SENSITIVITY TO DIGITALIS

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The average doses of digitalis required for the initial digitalization and subsequent maintenance of the heart patient are well known, but the averages do not indicate the range of individual differences encountered from one patient to another. Resistance to digitalis, and to the glycosides derived from it, is illustrated by the histories of four patients. It is sometimes impossible to give enough digitalis to slow down a tachycardia without inducing nausea, vomiting, pulsus bigeminus, or other signs of intoxication. Extreme sensitivity to digitalis is illustrated by the histories of three patients who exhibited intoxication with amounts of digitalis generally regarded as safe and usually given with impunity. Such sensitiveness has been seen after digitalis leaf, digoxin, digitoxin, and acetylstrophanthin. Awareness of the range of digitalis tolerance is essential if digitalis is to be used safely and effectively. Single-dose methods are hazardous. Digitalis should be given in divided doses, and the process of digitalization should be thought of as an individual biological titration.

The introduction of the purified glycosides has made available drugs that are standardized by weight and are of uniform potency. This standardization has popularized fixed dosage schedules both for digitalization and for maintenance therapy. Quantitative formulas have been proposed to predict digitalis requirements. These formulas are either based on the patient's weight or derived from statistical studies of the average requirements for large groups of cardiac patients. Even a single-dose technique for achieving digitalization has been advocated. Average dose formulas, as well as other dogmatic oversimplifications in prescriptions of digitalis drugs, disregard biological variability. Since individual requirements are unpredictable, single-dose methods of digitalization constitute a distinct hazard for the patient. At the present stage of our knowledge, clinical practice demands that digitalis be administered in divided doses. In effect, the process of digitalization constitutes a biological titration, during which the dose which will achieve the maximum therapeutic action is determined.[1]

The wide variability in digitalis dosage requirements is not generally appreciated. For example, in some patients a so-called digitalizing dose may prove inadequate for daily maintenance therapy,

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while in others a so-called daily maintenance dose may prove excessive for initial digitalization. The object of this report is to illustrate the extremes in digitalis requirements and to explore some of the factors which determine the variability in cardiac responsiveness. For this purpose seven patients have been selected for presentation out of a considerable number who have displayed similar features.

Resistance to Digitalis

The patients in this group showed little response to digitalis until amounts were administered which are generally regarded as adequate to produce intoxication. These large doses resulted in therapeutic action without evidence of toxicity. The following case reports demonstrate the problem of resistance, as well as some of the predisposing factors.

Case 1.—A 58-year-old man was hospitalized because of a ventricular rate of 240 beats per minute resulting from atrial flutter with 1:1 response. The first day he received 1.4 mg. of digitoxin intravenously and 0.6 Gm. of digitalis leaf orally without effect. During the following week an additional 4.6 Gm. of digitalis leaf was given. This slowed the ventricular rate to 118 beats per minute. Sinus rhythm was restored with quinidine. X-ray studies after reversion showed no cardiomegaly.

A year later 1:1 flutter recurred with a ventricular rate of 212 beats per minute. A total of 3.3 mg. of digitoxin given intravenously over a period of 60 hours was required to produce 2:1 atrioventricular block. Over the following 10 days an average of 0.6 mg. of digitoxin per day (range 0.4-0.8 mg.) was necessary to prevent ventricular acceleration. Reversion was again achieved with large doses of quinidine. He was then maintained with digitalis therapy.

A third episode of atrial flutter with a 1:1 response and a ventricular rate of 190 beats per minute occurred one and a half years later. Maintenance therapy with digitalis had been continued in the interval between paroxysms. Eleven milligrams of digoxin and 1.8 Gm. of digitalis leaf were required to control the ventricular rate during a 12-day period until a normal sinus mechanism was restored with quinidine. Half of the digoxin was given by the intravenous route. The ventricular rate was never less than 90 beats per minute. At no time were subjective manifestations of digitalis intoxication present. The patient has remained well for six years after this last episode and has been continued on a daily maintenance dose of 0.1 Gm. of digitalis leaf.

In this patient massive doses of digitalis failed to restore a sinus mechanism, produce atrial fibrillation, or control satisfactorily the ventricular rate. These large doses, however, slowed the ventricular rate sufficiently to permit a safer administration of quinidine. With the second episode of flutter, he received 9.3 mg. of digitoxin in 13 days. The dissipation of this preparation requires about three weeks. He was therefore under the influence of an amount of drug which ordinarily would have sufficed to produce digitalization in five or even six individual patients. Despite these large quantities, he exhibited none of the stigmata of digitalis overdosage. The resistance to digitalis was not conditioned by the type of medicament employed; he was equally unresponsive to digitalis leaf, digitoxin, and digoxin. The resistance was not occasioned by impaired gastrointestinal absorption, because parenteral administration did not enhance drug action. Thyrotoxicosis was presumably not a factor; thyroid function studies were normal.

Two questions are raised concerning the decisive tolerance for digitalis exhibited by this patient. First, why did 1:1 conduction persist and atrioventricular block result only after inordinately large doses—and even then the conduction defect was never greater than 2:1? Second, why did not digitalis intoxication develop? The persistent 1:1 atrioventricular conduction may have been due to a reduction in vagal tone. The fact that carotid sinus massage as well as prostigmine failed to evoke transient block supports this view. The control of the ventricular rate by digitalis is primarily indirect and medicated through the vagus nerve. This is an early effect of digitalis, produced with a small dose, and it may be annulled with atropine. If vagus activity is reduced or absent more of the drug is necessary for direct action of digitalis in prolonging the refractoriness of the atrioventricular conduction system.

The absence of digitalis intoxication may have been due to the state of the heart muscle. The capacity to withstand large doses of digitalis depends on the structural integrity of the myocardium.[1] When the heart is diseased, even small amounts may favor ventricular ectopic activity; conversely, when the heart muscle is normal, overwhelming doses may fail to produce such arrhythmias. There was no evidence of significant organic heart disease in this patient.

Case 2.—A 39-year-old woman with atrial fibrillation, a history of multiple cerebral embolic episodes, and resistant congestive heart failure was admitted for mitral valvuloplasty. During surgery she developed a cerebral embolus and did not regain consciousness. Shortly after the operation she developed atrial flutter with an atrial rate of 350 beats per minute and a 2:1 ventricular response. Her temperature was 104 F (40 C). She was on maintenance therapy with digitoxin, and an additional dose of 0.5 mg. of digoxin was given intravenously without effect. Since her state of digitalization was uncertain, a digitalis tolerance test was carried out. Within 40 minutes after she received 2.4 mg. of acetylstrophanthidin given intravenously, atrial fibrillation with a slow ventricular rate was restored. She continued to have a high fever, and she died the next day. A postmortem examination revealed a basilar artery embolus with softening of the internal capsule and infarction of the pons.

Prior to surgery, 0.1 mg. of digitoxin daily sufficed to maintain a slow ventricular rate. With the emergence of atrial flutter a large additional intravenous dose of digoxin as well as nearly twice the usual digitalizing dose of acetylstrophanthidin was required to reestablish atrial fibrillation. This patient again demonstrates the resistance to digitalis which may be encountered in the presence of atrial flutter. Two more factors may have been operative. The requirements of digitalis increase with febrile reactions.[2] It is our impression that more digitalis

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frequently is necessary to control a cardiac arrhythmia occurring in association with a cerebrovascular accident.

Case 3.—A 35-year-old woman with congestive heart failure due to mitral stenosis had been maintained on 0.1 Gm. of digitalis leaf daily for three years. Within 24 hours after a successful mitral valvuloplasty, atrial fibrillation developed for the first time. The ventricular rate varied around 175 beats per minute. This rapid rate was not affected by supplementation of the daily maintenance dose with 1.0 mg. of intravenous digoxin on two successive days. Digitoxin in an amount of 0.3 mg. per day for three days was also without effect. The rate was finally lowered to 105 per minute after an additional dose of 1.2 mg. of digitoxin was given in the course of three days. She required a maintenance dose of 0.3 mg. of digitoxin daily. On the 14th day after operation sinus rhythm was spontaneously reestablished. Thereafter, 0.1 mg. of digitoxin proved adequate. During the two weeks after operation the blood urea nitrogen level, hematocrit, and serum electrolytes remained unaltered.

When a digitalized patient develops atrial fibrillation, it is frequently necessary to increase the dose of digitalis. This is especially true when atrial fibrillation begins after mitral valve surgery. The need for increased digitalis dosage may be due to the fact that many patients with a normal sinus mechanism are incompletely digitalized. In the presence of atrial fibrillation, the ventricular rate serves as a sensitive indicator of the state of cardiac compensation. The rate does not constitute an index when sinus rhythm prevails. Thus, if a patient is under-digitalized, ventricular acceleration would ensue with the development of atrial fibrillation. It is also possible that the factors responsible for atrial fibrillation burden the cardiac reserve, increase the degree of decompensation, and thus raise digitalis requirements. These explanations do not account for the resistance to digitalis encountered occasionally after mitral valvuloplasty. After surgery in patients who have established fibrillation, the ventricular rate may be more rapid and not yield as well to digitalis therapy. In these instances a number of factors may be implicated, including trauma to heart muscle during surgery, febrile reaction, pulmonary atelectasis and inflammation, deranged electrolytes and water distribution, cerebral anoxia, and perhaps even the elaboration of adrenal cortical steroids that compete biologically with the group of digitalis steroids.

Case 4.—A 42-year-old woman with mitral stenosis, mitral insufficiency, aortic insufficiency, and moderately severe essential hypertension redeveloped cardiac decompensation three years after undergoing mitral valvuloplasty. Atrial fibrillation had been present for four years. Though she received 0.2 Gm. and 0.3 Gm. of digitalis leaf on alternate days, her ventricular rate was 120 to 130 beats per minute. Comparable amounts of orally given digitoxin were without effect in lowering the heart rate. When, in addition to maintenance doses of digitalis, 6.5 mg. of digoxin was given orally over a period of five days, gastrointestinal upset was produced without material rate reduction. A dose of 0.3 mg. of acetylstrophanthidin given intravenously resulted in slowing of the rate to 88 beats per minute, but bigeminal rhythm developed. On two occasions when she was without digitalis for two days, 0.5 mg. of intramuscularly administered digoxin slowed the rate to 85 beats per minute. This was accompanied by nausea and vomiting. There was no evidence of active rheumatic carditis. Acute phase reactants were negative. Hydrocortisone given intravenously did not render the rate more susceptible to slowing by digitalis. She sweated frequently, ran a low-grade fever, felt flushed, and tolerated hot weather poorly. However, the thyroid gland was not enlarged, the radioiodine uptake was 25%, the basal metabolic rate was +16%, and the serum protein-bound iodine level was 6.4 mcg. per 100 cc.

The heart rate was not slowed by supplementation of the large daily maintenance dose of digitalis with orally given digoxin (Lanoxin). The amount of additional digoxin would have sufficed to accomplish fully digitalization in a patient with cardiac disease who had never received digitalis. Gastrointestinal upset precluded administration of still larger doses of the drug. On three occasions much smaller doses of parenterally administered digitalis produced slowing of the heart rate. These were accompanied by either subjective or objective manifestations of overdosage of digitalis. The reason for the greater effectiveness of the parenteral route is that the orally administered drug may not have reached the heart. This may have been due to either impaired gastrointestinal absorption or more complete hepatic inactivation. Studies with radioactive digitoxin have demonstrated that the liver is the main organ concerned with its detoxification.[3] Ingested digitalis presents a higher concentration to the liver than with the parenterally administered drug, thus permitting hepatic inactivation with a lesser fraction of the active principle reaching the heart.

Sensitivity to Digitalis

In the category of sensitivity to digitalis, selected patients are considered who exhibited intoxication with amounts of digitalis generally regarded as safe and usually given with impunity.

Case 5.—A 66-year-old woman with multivalvular rheumatic disease was hospitalized because of cardiac arrhythmia and increasing heart failure. An increase in digitoxin dose to 0.3 mg. for one day resulted in nausea, vomiting, palpitation, and increasing shortness of breath. On admission to the hospital she showed cachexia and was critically ill. There was marked cardiomegaly with evidence of heart failure on both right and left sides. The cardiac rate was 44 beats per minute; it was regular, with variable intensity of the apical first sound. Electrocardiograms confirmed the presence of complete heart block but demonstrated in addition an atrial tachycardia with a rate of 186 beats per minute. Administration of procainamide hydrochloride and potassium, as well as withdrawal of digitalis, resulted in slow resolution of the arrhythmia.

For 13 days she received no digitalis. During this time she developed atrial fibrillation. Redigitalization with oral administration of 1.0 mg. of digoxin given in fractional doses in the course of a single day caused recurrence of nausea and vomiting. Digitalization was therefore halted and maintenance therapy initiated with a dose of 0.25 mg. of digoxin daily. The frequent complaint of gastrointestinal upset caused numerous alterations in digitalis dosage. There was a progressively increasing intolerance to digitalis medication. Even 0.25 mg. of digoxin every third day provoked nodal

rhythm and bigeminy with multiform ventricular beats. A number of physicians believed that the various manifestations ascribable to digitalis were, in fact, due to advancing decompensation and underdigitalization. Therefore an acetylstrophanthidin tolerance test was carried out. At the time of the test she had had no digoxin for 10 days. The underlying rhythm was atrial flutter. Within five minutes after the first increment of 0.15 mg. of acetylstrophanthidin, bigeminal rhythm developed. This dose is but a small fraction of the amount usually necessary to initiate digitalization. Since the development of bigeminy may have been fortuitous, the test was repeated nine days later. At this time she had received no digitalis medication for 19 days. Again bigeminal rhythm recurred after 0.15 mg. of acetylstrophanthidin was administered.

When the digitalis dose was increased for one day severe intoxication occurred which lasted for four days, notwithstanding the administration of potassium and procainamide.[4] Thereafter, the sensitivity to digitalis was such that effective therapy could no longer be carried out. Even a single dose of 0.25 mg. of digoxin every two or three days was not tolerated. This sensitivity was not limited to orally administered digoxin but also was apparent and even more striking after intravenous administration of acetylstrophanthidin.

What was the basis for the marked sensitivity to digitalis? The most likely explanation is that the severe heart disease caused the digitalis intolerance. However, in this case increased sensitivity developed abruptly after the episode of digitalis intoxication. Prior to this, the patient had received 0.2 mg. of digitoxin in daily doses for a year without untoward effect. It is possible that an overdose of digitalis, in the presence of advanced heart disease, induced permanent biochemical damage to the myocardium. Digitalis in overdose causes loss of potassium from the heart muscle.[5] Such loss may not be restored when the myocardium is continuously subjected to anoxia, even when the agent initiating the depletion is withdrawn. A deficit of myocardial potassium would result in an augmented sensitivity to digitalis.[1]

An alternate explanation is that digitalis was present in the body during the final episode of illness, which lasted four months. Digitoxin is generally thought to persist in the body for three to four weeks. Unchanged drug has been found in the urine for as long as 50 days after digitoxin biosynthetically labeled with radiocarbon (C^{14}) has been given.[6] In dogs depleted of potassium the duration of toxicity resulting from acetylstrophanthidin, which usually remains in the body for less than 15 minutes, was prolonged for many hours. It may be that in severe cardiac decompensation failure of digitalis detoxification by the liver prolongs its period of action, and this in turn maintains myocardial potassium depletion. The clinical result would be an extreme degree of long-lasting digitalis intolerance.

Case 6.—A 60-year-old robust man with rheumatic aortic valvular insufficiency and cardiomegaly, but without heart failure, was given maintenance doses of digitalis to prevent recurrences of bouts of atrial tachycardia. He was never digitalized with a long-acting preparation but was given a maintenance dose of digitoxin, 0.2 mg. four times a week. After several months he experienced nausea and anorexia. The sinus rate was 55 per minute, and he exhibited occasional ventricular premature beats. The dose was reduced, therefore, to 0.1 mg. per day for four and then for three days a week. However, he began to complain of being "abstracted" several times each day. He found it difficult to elaborate on this symptom. His associates and family explained it as a momentary lapse of consciousness with transient periods of disorientation. This very sensation of "abstractness," as well as cardiac standstill, was reproduced by brief carotid sinus massage. After digitalis therapy was stopped, carotid sinus stimulation evoked neither symptoms nor cardiac standstill.

A patient may be ultimately digitalized by only a maintenance dose given daily, even though he has never received larger doses before. In the course of several weeks, enough of the drug accumulates in the body to produce its effect. Under these circumstances this daily dose is slightly larger than the maintenance requirements for that particular person. In fact, digitalis intoxication can result from such a procedure, as occurred in the patient described. The sensitivity in this patient was primarily due to increased vagal effect on the sinus node. It is well established that digitalis preparations may sensitize the carotid sinus predisposing to syncopal attacks. As little as 3 grains (0.2 Gm.) of digitalis leaf may be accompanied by increased carotid sinus sensitivity.[8] In this patient, substituting quinidine for digitalis prevented the bouts of rapid heart action and abolished the carotid sinus sensitivity. An alternate approach might have been the use of atropine in conjunction with digitalis. Since the sole reason for digitalis, however, was to prevent recurrence of paroxysmal atrial tachycardia, it seemed more advisable to switch to quinidine rather than manipulate two medications.

Case 7.—The patient was a 43-year-old woman with multivalvular heart disease—mainly aortic stenosis, atrial fibrillation, and cured subacute bacterial endocarditis. An aortic valvuloplasty resulted in objective improvement. Both before and after surgery frequent attacks of rapid heart action were not readily controlled by digitalis. Because of laboratory findings suggesting thyrotoxicosis, she was given two doses of radioactive iodine, totalling 18 mc. Ten weeks after receiving antithyroid medication, she was rehospitalized because of repetitive paroxysms of rapid ventricular rates ranging from 175 to 200 beats per minute. The rhythm continued to be atrial fibrillation. Small supplements of digoxin controlled the arrhythmia transiently but evoked either complete heart block or bigeminal rhythm. Digoxin in a dose of 0.25 mg. every second or third day proved excessive. The ventricular rate varied from 40 to 190 beats per minute. The protein-bound iodine level was found to be 1.2 mcg. per 100 cc. After thyroid replacement therapy she tolerated 0.15 mg. of digitoxin daily without manifesting arrhythmias. She still had heart rates up to 130 beats per minute but without periods of excessive bradycardia.

In this unusual case, atrial fibrillation with a rapid ventricular rate could not be controlled readily by digitalis without provoking digitalis intoxication. The basis for the paroxysms of rapid heart action remains unexplained. One might have expected that lowering thyroid function would have lessened or eliminated the paroxysms. However, this was not the case. In effect, marked sensitization to digitalis occurred, so that it could not be employed effectively without provoking intoxication. Once adequate thyroid replacement was instituted the arrhythmia could be managed more readily by digitalis therapy.

Comment

Determination of the optimal dose of a digitalis preparation for any given patient continues to be an important problem in the management of congestive heart failure. For a large group of patients a dosage response curve, as well as a dosage toxicity curve, can be formulated, indicating the amount of drug which is most likely to produce the maximum therapeutic and the least toxic effect. However, in any particular patient, at the present time, accurate prediction of this dose is not possible. Proper therapy demands awareness of the extremes of individual variability. The difference in patients who present cardiac problems may be very marked. This is particularly evident when cases 1 and 5 are compared. In the former, even after the patient was assumed to be fully digitalized, huge additional doses resulted in therapeutic but no toxic action. In the latter, single doses of 0.25 mg. of digoxin evoked toxic effects even when it could be assumed that the patient was not at all digitalized.

In the majority of patients no such extremes are encountered. It is therefore possible to digitalize most patients with safety by means of small, properly spaced, fractional doses. Nevertheless, the problem is found in the occasional patients in whom routine digitalization would endanger the ultrasensitive individual while it would not give therapeutic benefit to the individual with exceptional resistance.

What are some of the factors producing the extremes in responsiveness? The key factor is the state of the myocardium. In the absence of heart disease large doses may be taken without ill-effect. This is well illustrated in the reports of attempted suicide with digitalis drugs. Bickel [10] reported a suicidal attempt by a 36-year-old woman without heart disease who consumed a dose of 50 mg. of digitoxin (Digitaline Nativelle). The drug was in liquid form and was diluted with warm water. Nausea, vomiting, somnolence, resistant hiccups, visual manifestations, and severe hypotension followed. The electrocardiogram revealed changing atrial pacemaker, paroxysms of atrial flutter and fibrillation, and first degree heart block, the latter persisting for 11 days. No alterations in the ventricular complex were observed. The patient made an uneventful recovery. The sparing of the ventricle after massive doses of digitalis in the absence of heart disease has been also noted by others.[11] When the heart is normal, ventricular abnormalities due to digitalis are unusual, and if they occur they are of brief duration. In the presence of heart disease, ventricular ectopic rhythms are common and readily provoked by digitalis. With increasing severity of heart disease, the margin between the therapeutic and toxic doses progressively decreases. At times no beneficial action can be obtained without toxic effects. In the terminal stages of decompensation, toxicity may precede therapeutic action and it may be impossible to use digitalis profitably.

Next in importance in determining the myocardial threshold for digitalis is the state of body electrolyte distribution. Electrolyte and water abnormalities accompany heart failure. These are due to both derangement in heart function and the therapeutic procedures in current use. While calcium plays a significant role in modifying the action of digitalis, in clinical practice the key ion is potassium. Depletion of potassium, no matter how produced, sensitizes the heart; under such circumstances even small doses of digitalis may result in advanced intoxication.[4] Depletion of potassium in animals with normal hearts lowers digitalis requirements to the same degree as does cardiac decompensation in animals without potassium deficits.[7] It may be that the reduced threshold to digitalis observed with advanced heart failure is in some measure due to deficits of myocardial potassium.

Sensitivity to digitalis may also be encountered in patients with acute myocardial infarction, acute cor pulmonale, and rheumatic carditis, during calcium administration and adrenal steroid therapy, in the presence of acute renal shutdown, in the terminal stages of uremia, after large infusions of glucose, and in the very aged. Whether the common denominator for digitalis sensitivity in this heterogenous group is an electrolyte disturbance within the myocardium is uncertain at present.

A number of factors or states tend to increase digitalis requirement, though this does not always go hand in hand with increased resistance to the toxic effects. Following are some of the situations: in the presence of thyrotoxicosis, febrile illness, myocarditis, impaired gastrointestinal absorption, or more rapid and complete hepatic inactivation; occasionally after valvular heart surgery; and in infants under the age of 2 years.[8] Other conditions in which resistance is thought to occur at times include pneumonia (especially lobar), pneumothorax, after pulmonary embolic episodes, in atrial fibrilla-

tion developing in patients with Wolff-Parkinson-White syndrome, in beriberi heart disease, in the presence of a ball-valve thrombus in the left atrium, and in arrhythmias resulting from metastasis to the myocardium.

Awareness of the role of the above enumerated factors permits treading with caution when sensitivity is suspected. This helps in avoiding serious intoxication. When resistance is anticipated, it permits the use of digitalis with boldness to assure full therapeutic action. This, at times, may prove life-saving.

Summary

There is great variability in the amount of digitalis necessary for optimum effect in different individuals. In a considerable group of patients unusual resistance or marked sensitivity may occur.

The two most important factors which determine the responsiveness to digitalis are the state of the heart muscle and the electrolyte balance in the body. With increasing severity of cardiac decompensation, especially when this is the result of heart muscle insufficiency, smaller doses of digitalis will produce intoxication. When body potassium is depleted, enhanced sensitivity to digitalis results. The operation of these two factors may be the common underlying basis for sensitivity as well as resistance to digitalis under all circumstances.

Awareness of these principles is of clinical significance. At times it will prevent severe digitalis intoxication and even fatality. At other times it will enable one to attain cardiac compensation and recovery when the condition might otherwise be regarded as refractory to therapy.

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MULTIPLE ANTIGEN IMMUNIZATION.—Two groups of infants received poliomyelitis immunizations beginning at the age of 6 weeks and completing the series by the age of 10 months. One group was given the vaccine as a separate injection while the other received the vaccine mixed in the same syringe with pertussis or diphtheria-pertussis-tetanus vaccine. The responses of these infants to poliomyelitis vaccine were analyzed and the following general conclusions were reached: Poliomyelitis vaccine administered to infants as young as 6 weeks, and given according to the usually recommended schedule, results in a satisfactory antibody response. The response to poliomyelitis vaccine mixed with pertussis and DPT vaccines and given as one injection is satisfactory. The combination of antigens gave no practical interference to development of antibodies to diphtheria and pertussis. There were no apparent hazards or adverse reactions associated with combined poliomyelitis-pertussis or poliomyelitis-DPT vaccines. There were no differences in poliomyelitis antibody responses following immunizations with separate and combined vaccine injection. The existence of maternal antibodies at the time of poliomyelitis immunization had no apparent influence on the production or persistence of active antibodies. These findings indicate that poliomyelitis immunization procedures can very easily be integrated into routine pediatric immunization programs, thereby affording the earliest possible protection.—R. Batson, M.D., A. Christie, M.D., B. Mazur, M.D., and J. H. Barrick, B.A., Response of the Young Infant to Poliomyelitis Vaccine Given Separately and Combined with Other Antigens, *Pediatrics*, January, 1958.

EVALUATION OF TES-TAPE AS A QUANTITATIVE INDICATOR

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Paper strips impregnated with an enzyme, glucose oxidase, can be used to test for the presence, and also to estimate the concentration, of glucose in urine. The accuracy of such estimates was investigated by testing 104 samples made by adding predetermined amounts of glucose to normal urine to give concentrations of 10, 5, 3, 2, 1, 0.5, 0.25, and 0.1%. The test discriminated correctly among these possibilities except in six cases, four 0.25% samples being misread as 0.5% and two 0.1% samples being misread as 0.25%. The effects of varying the pH, of letting the urine stand at different temperatures, of contaminating it with bacteria, and of exposing the test strips to water, air, and sunlight were studied, as were the responses of the strips to solutions of other sugars (maltose, fructose, and galactose). The test was found to be specific for glucose. The enzyme must be protected against the destructive action of heat, air, and light. Used with due care, especially with respect to timing, the test discriminates reliably among the above-mentioned concentrations of glucose in urine and is a practical method for observing the course of clinical glycosurias.

There has been some disagreement reported as to the reliability of Tes-Tape (strips impregnated with the enzyme, glucose oxidase) in the quantitative determination of urinary sugar. Results reported by Comer [1] indicate that Tes-Tape had an over-all accuracy of 98% in the testing of 1,500 urine samples with glucose concentrations of 0 to 2%. Leonards,[2] on the other hand, found Tes-Tape satisfactory as a quantitative indicator only for testing urine containing 0.25% glucose or less. Urine specimens that contained 0.5% glucose or more were often found to give readings that were deceptively low. Since a rapid, easily performed, quantitative examination for urinary sugar is of obvious value to the patient, the physician, and the laboratory, a study was undertaken to re-evaluate the reliability of Tes-Tape in quantitative determination. Special emphasis was placed on factors, affecting both the paper strips and the urine, that might lead to erroneous results.

All tests, with the exception of one series of unknowns, were performed by one laboratory worker and were performed exactly in accordance with the furnished instructions. Normal urine specimens that had been collected from hospital patients and found to be free of sugar or other abnormal constituents were used for all studies in which glucose concentrations were prepared. Urine samples collected from diabetic patients were used in one series of tests. Bacterial cultures used were isolated from hospitalized patients having urinary tract infections.

Tests and Results

Urine and distilled water solutions containing mannose, inulin, lactose, xylose, sucrose, fructose, galactose, and maltose were tested with Tes-Tape and gave no positive results in concentrations lower than 5%. Galactose gave a reading of 0.1% at the 5% concentration, and maltose gave a reading of 0.1% at the 10% concentration. Fructose produced a slight color change at 5% and at 10% concentrations. Since high concentrations of these sugars are not likely to be found even in abnormal urine, the Tes-Tape strips can be considered specific for glucose determination. The results of this comparison of the readings obtained using the sugars mentioned are shown in table 1. It should be noted that "2% or more" is the highest concentration which may be determined by use of Tes-Tape. In all results reported, 2% should be interpreted as meaning 2% or more.

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To evaluate Tes-Tape in quantitative determination, 104 urine samples containing varying concentrations of glucose were tested. Glucose concentrations of 10, 5, 3, 2, 1, 0.5, 0.25, and 0.1% were used. The two laboratory workers performing the study had previously familiarized themselves with the technique of the test, but the concentration of glucose in each urine sample was unknown to them. Of the 104 specimens examined, 6 were read in-

TABLE 1.—*Comparison of Results Obtained with Tes-Tape Used for Detection of Various Sugars in Urine**

	Concentration, %								
	10	5	3	2	1	0.5	0.25	0.1	0.01
Glucose	2†	2	2	2	2	0.5	0.25	0.1	0
Maltose	0.1	T‡	T	0	0	0	0	0	0
Fructose	T	T	0	0	0	0	0	0	0
Galactose	0.25	0.1	T	T	0	0	0	0	0

* Mannose, inulin, lactose, sucrose, and xylose gave negative results in all concentrations.
† Values of 2 indicate 2% or more—highest reading possible with Tes-Tape color chart.
‡ T: less than 0.1% but a definite color change.

correctly. Four of these contained 0.25% glucose and were read as 0.5%. Two urine samples containing 0.1% glucose were read as 0.25%. Nine of the specimens examined had a glucose level of 1%, and of these four were recorded as containing 0.5% glucose and five were recorded as containing 2% glucose. Since the color chart provided with the Tes-Tape strips does not include a color standard for 1% glucose, these latter nine results cannot be considered incorrect but are inconsistent. Contrary to the results obtained by Leonards, in no concentration for which a color standard was provided was a reading made that was lower than the true glucose level in the urine tested.

Variation in pH was considered as a possible source of error in the use of enzyme glucose oxidase strips. Normal urine with glucose added to give concentrations of 10, 5, 3, 2, 1, 0.5, 0.25, and 0.1% were used and the pH adjusted to pH 2 to pH 12 for each glucose concentration. Results of tests with use of Tes-Tape proved the method to be accurate in the range of pH 4 to pH 9. At pH levels below 4 and above 9 urine samples containing glucose concentrations of 0.1% gave readings of 0.25%. It was found that, at each extreme of the pH range used, the color change appeared only in a narrow zone at the solvent line. As reported by Comer, ascorbic acid in concentrations above 0.05% was found to retard color formation at the 0.1% glucose level.

We examined the effects of standing, using urine specimens from diabetics. These specimens contained no bacterial contamination and gave a 4+ reading by Benedict's method and a Tes-Tape reading of 2% or more. The specimens were divided and held for 48 hours at 37 C and at room temperature and were tested at irregular intervals during this time. Standing at either temperature appeared to have no effect on the results of Tes-Tape determination.

We considered the effect of growth in urine—due to urinary tract infections or to outside contamination of urine samples held for any length of time—as a source of error in the use of the enzyme strips. Normal urine specimens with glucose added to give concentrations of 10, 5, 3, 2, 1, 0.5, 0.25, and 0.1% were seeded with 18-hour broth cultures of Pseudomonas aeruginosa, Proteus vulgaris, Escherichia coli, Staphylococcus pyogenes var. aureus, and hemolytic streptococci. Duplicate tests were made at 37 C and 4 C, and readings were recorded at the end of 24, 48, and 72 hours. It was found that the urine specimens seeded with all organisms tested and incubated at 37 C gave negative readings at the 0.1% glucose level in 24 hours. The strain of P. vulgaris used utilized the largest amount of glucose, as indicated by a reading of 0.5% at the glucose level of 2% after 24 hours at 37 C. The results of the tests made on urine held at 4 C showed that even at this temperature P. vulgaris, Ps. aeruginosa, Esch. coli, and Staph. aureus were capable of metabolizing enough glucose to give a negative reading in 48 hours at the 0.1% glucose level. The presence of hemolytic streptococci did not appear to alter the readings at the end of 72 hours. Results of this study are shown in tables 2 and 3.

The ability of the test paper to withstand various types of abuse was next taken into consideration. Tes-Tape strips moistened with tap water and exposed to circulating air at room temperature for 24 hours were found to produce a color change but were inaccurate for determining varying concentration of glucose solutions. Strips moistened with tap water and dried for three minutes at 100 C were completely inactivated. Exposure to ultraviolet light for 1, 3, and 10 minutes or to direct sunlight for one hour did not affect the accuracy of readings at various glucose levels. Exposure to

TABLE 2.—*Effects of Bacterial Contamination on Results with Tes-Tape in Urine Specimens Incubated at 37 C*

	Glucose Concentration, %																					
	10			5			3			2			1			0.5			0.25			0.1		
Incubation time, hr	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72
Normal urine and																								
Ps. aeruginosa	2*	2	2	2	2	2	2	2	0.5	2	0.5	0.25	0.5	0.25	0	0.25	0.1	0	0.1	0	0	0	0	0
P. vulgaris	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0.25	0.25	0	0	0	0	0	0	0	0	0	0	0
Esch. coli	2	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.25	0.1	0.25	0	0	0	0	0	0	0	0
Staph. aureus	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0	0	0.25	0	0	0.1	0	0	0	0	0
Beta-hemolytic streptococci	2	2	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0.25	0	0	0.1	0	0	0	0	0

* Values of 2 indicate 2% or more—highest reading possible with Tes-Tape color chart.

TABLE 3.—*Effects of Bacterial Contamination on Results with Tes-Tape in Urine Specimens Incubated at 4 C*

	Glucose Concentration, %																							
	10			5			3			2			1			0.5			0.25			0.1		
Incubation time, hr	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72	24	48	72
Normal urine and																								
Ps. aeruginosa	2*	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0.25	0.25	0.25	0.1	0.25	0.1	0	0	0	0
P. vulgaris	2	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0.5	0.25	0.1	0.1	0	0	0	0	0	0
Esch. coli	2	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0.5	0.25	0.1	0	0.1	0	0	0	0	0
Staph. aureus	2	2	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0.5	0.5	0.1	0.25	0.1	0.1	0.1	0	0
Beta-hemolytic streptococci	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	0.5	0.5	0.5	0.25	0.25	0.25	0.1	0.1	0.1

* Values of 2 indicate 2% or more—highest reading possible with Tes-Tape color chart.

direct sunlight for periods longer than one hour produces a gradual decrease in the sensitivity of the strips. Table 4 shows the results obtained from this series of studies.

Comment

Although solutions of fructose, galactose, and maltose produce some color change in high concentrations when tested with Tes-Tape, the strips may be considered specific for glucose at the sugar levels found even in abnormal urines. The pH range in urine does not affect the accuracy of the readings made with Tes-Tape strips. This indicates a wide pH stability of the enzyme. Results of quantitative determinations made with use of Tes-Tape indicate the accuracy of the test to be 94% or higher, and the incorrect results were in each case higher than the true glucose level in the urine tested. It was found to be essential that the darkest zone of color be used in making a reading and that examination of the strips be made as nearly as possible at exactly one minute. Accuracy in recording results was found to depend to a great extent on careful timing. It was also found that experience in performing the test increased the accuracy of the results obtained. Standing did not appear to affect the results obtained unless the urine was contaminated. In cases of urinary tract infections it was found to be essential that the urine not be allowed to stand at room temperature longer than was necessary before testing for urinary sugar. Urine specimens containing bacterial contamination were not satisfactory for examination if held for as long as 24 hours at refrigerator temperature. Bacteria which are most often found in urinary tract infections utilize the glucose in urine to the extent that negative or deceptively low results may be obtained by any method used for glucose determination. The Tes-Tape strips were found to be stable under conditions normally encountered in the laboratory if protected from excessive heat, wetting, and extended exposure to direct sunlight. Since Tes-Tape quantitative sugar determination requires no preliminary preparation and takes only slightly over one minute to perform, it has a decided advantage over more time-consuming methods when heavily contaminated urine specimens are examined.

Table 4.—*Results of Exposure to Environmental Factors Affecting Stability of Tes-Tape Strips*

	Glucose Concentration, %							
	10	5	3	2	1	0.5	0.25	0.1
Tape wet with tap water and air-dried	0.5	0.5	0.5	0.5	0.5	0.25	0.25	0.25
Tape wet with tap water and dried at 100 C for 3 min.	0	0	0	0	0	0	0	0
Exposure to ultraviolet light								
1 min.	2*	2	2	2	2	0.5	0.25	0.1
3 min.	2	2	2	2	2	0.5	0.25	0.1
10 min.	2	2	2	2	2	0.5	0.25	0.1
Exposure to direct sunlight								
1 min.	2	2	2	2	2	0.5	0.25	0.1
5 min.	2	2	2	2	2	0.5	0.25	0.1
30 min.	2	2	2	2	2	0.5	0.25	0.1
1 hr.	2	2	2	2	2	0.5	0.25	0.1
2 hr.	2	2	2	2	0.5	0.5	0.25	T
4 hr.	2	2	2	2	0.5	0.5	0.1	0
6 hr.	T†	T	T	0	0	0	0	0

* Values of 2 indicate 2% or more—highest reading possible with Tes-Tape color chart.
†T: less than 0.1% but a definite color change.

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CORTISONE AND POLYARTERITIS NODOSA.—All instances of rheumatoid arthritis examined at necropsy at the Mayo Clinic through 1954 were reviewed, with special emphasis on the incidence and character of the vascular lesions and their relationship to the administration of cortisone. There were 52 patients in the study. Fourteen of these had received cortisone and 38 had not. Four (29%) of the group of 14 treated with cortisone had generalized lesions of polyarteritis nodosa, whereas none of the 38 patients who did not receive cortisone had such lesions. These findings suggest that, in certain susceptible patients with rheumatoid arthritis, the administration of cortisone may precipitate the development of diffuse necrotizing arteritis. The explanation as to why this may occur in some patients with rheumatoid arthritis treated with cortisone and apparently not in patients with other diseases (with the possible exception of disseminated lupus erythematosus) is not clear. . . . We found no vascular lesions specific for rheumatoid arthritis in this study.—J. W. Kemper, M.D., A. H. Baggenstoss, M.D., and C. H. Slocumb, M.D., The Relationship of Therapy with Cortisone to the Incidence of Vascular Lesions in Rheumatoid Arthritis, *Annals of Internal Medicine*, May, 1957.

CERTIFIED CAUSE OF DEATH—ANALYSIS AND RECOMMENDATIONS

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Discrepancies in the interpretation of findings reported as causes of death have often had serious consequences. The following material must be considered: history (past and present), the last illness, agonal phenomena, and autopsy (including all gross and laboratory data). Cases are cited showing that the omission of any one source of information can lead to a completely false conclusion. The mechanisms causing death are often multiple, complex, and difficult to analyze; pathogenesis may not parallel significance. An appropriate single listing of causation may thus be impossible. Terminal undemonstrable physiological factors can be important and quite independent of demonstrable dramatic positive findings. Provision for correction of certificates should be made on the basis of additional data. Consultation with an expert in certification or with a responsible authority before completion of the certificate is recommended. This routine would often clarify jurisdiction at the outset, would give diagnoses in keeping with acceptable nomenclature, and would enhance the accuracy of vital statistics. It would reduce the danger of injustice and spare the physician the embarrassment and irritation that follows an erroneous report.

The opinion of a physician as to the cause of death is written on a death certificate without mention of the background of facts substantiating the opinion. Sometimes haste and expediency influence his decision. Because of the wide range of error that obtains in determining the cause of death and in obtaining the written record of the facts under present certification procedure, a careful routine and all possible safeguards are desirable.

Medicolegal Classificaion

To attain a better scientific approach in determining the actual cause of death, the medicolegal classification of deaths is here listed, modified from that of Moritz.[1] Natural deaths include (1) instantaneous death, (2) sudden death (unexpected syncope with fatal unconsciousness or rapidly fatal illness with early prostration, without initial unconsciousness), and (3) expected death (prolonged illness, prolonged coma, or expected death with sudden termination). Unnatural deaths include (1) accident, (2) suicide, and (3) homicide (murder, with and without premeditation, or manslaughter in various degrees, both justifiable and technical).

For correct classification of a death, the actual facts as to its underlying cause must be established. Some cases in which miscarriage of justice may occur are cited here.

Case 1.—An indictment was drawn maintaining that a father "wilfully and maliciously drowned his child by holding the mouth under the faucet until she was dead." The autopsy protocol listed morphologic findings which did not justify the diagnosis of drowning. The chloride test for drowning was negative. A massive subdural hemorrhage confirmed the father's contention that the child had fallen accidentally. An assistant medical examiner stubbornly maintained, despite evidence to the contrary from his own protocol, that the cause of death was due to drowning alone, with no contributory cause. This discrepancy in interpretation is, in fact, due to a lack of a clear understanding of the meaning and intent of the phrase "cause of death." This phrase is not uniformly or clearly defined.

Case 2.—A young officer was charged with murder, because he was seen in the company of a housemaid shortly before she was found with a scalp wound, fracture of the skull, and intracranial hemorrhage. The testimony of several pathologists was required to free this man from the charge of striking a fatal blow, because the microscopic examination showed that the deceased person had been suffering from active and extensive syphilitic arteritis of the cerebral vessels with rupture and hemorrhage.

Case 3.—A husband was suspected of killing his wife in an altercation overheard by neighbors. It was found that the cause of her death was due to an active syphilitic disease of the blood vessels with actual gumma, accounting for the massive subarachnoid hemorrhage.

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Case 4.—A middle-aged man fell from a horse and was picked up dead. The basis for double indemnity seemed obvious, but the autopsy showed that a coronary occlusion was responsible for the fall and death.

Case 5.—A middle-aged man fell from a horse, but got up and continued his duties. Several months later, he went into coma and died rapidly. A tentative clinical opinion of apoplexy was offered, but the autopsy showed chronic subdural hemorrhage, undoubtedly related to the original trauma.

The consequences of discrepancies in the interpretation of findings can be serious in criminal and civil cases. Careful determination of the cause of death requires consideration of the following material: 1. History, including data obtained from family physician or medical advisor. 2. Clinical picture of the present illness, including symptoms, and laboratory data relative to the bacteriological, chemical, toxicological, and biopsy findings. 3. Clinical picture of agony, important in determining the terminal physiological mechanisms of death. In

New York City, visiting the scene is required of the medical examiner in cases of sudden death. Evaluation of the surroundings and confirmed statements from onlookers, friends, or relatives constitute important circumstantial evidence. 4. Autopsy. This is most important in establishing actual cause of death. It should include careful, complete gross dissection and microscopic studies and chemical, bacteriological, and viral data if warranted by findings and circumstances.

Generally, the clinical opinion is entered on the death certificate and is based on only the first three sources listed. Each case requires individual consideration, with evaluation of all data compiled from every available source. In one instance, a medical examiner certified coronary arteriosclerosis as the cause of death after a partial autopsy. Often, this is an acceptable cause of death. However, in this instance the deceased person had a history of trauma to the head, with an interval suggesting subdural hematoma. He went into coma and died after a period of time during which he showed neurological symptoms. Had this history been available to the medical examiner, it would have been clear that the head must be explored before a cause of death from this source could be excluded. Testimony subsequently demonstrated that the autopsy had been inadequate and that the stated cause of death was unjustified.

Relation of Demonstrable and Undemonstrable and Natural and Unnatural Factors in Mechanism of Death

In numerous cases, the cause of death is difficult to determine even when careful history, clinical observation, laboratory data, and autopsy results are available. Of course, the percentage of error increases when some or all of these data are unobtainable. As revealed by autopsy procedure, the range of error is 10% in the hands of able clinicians possessing laboratory aids. This parallels the experience of others.[2] The percentage of error when data from several of these sources, including data from autopsy, are not available is such as to diminish the validity of mortality statistics based on such material. A recent "Select Bibliography of Studies Relating to Accuracy of Cause of Death Statistics" [3] covers both precision of diagnosis and dependability of reporting procedure.

Assumption that autopsy will invariably establish the exact nature of the cause of death is unwarranted. In a considerable number of cases, the cause cannot be established by autopsy alone; also, there remains a group of deaths in which autopsy and all other data do not prove adequate to determine the actual cause of death. This pertains particularly to sudden deaths of a functional nature[4] and to some instances of poisoning or obscure intoxication. The gross autopsy is also often inadequate in cases involving vascular and viral diseases, which often do not show extensive morphologic alteration. These examples may illustrate.

Case 6.—A man bent over, compressed a hyperirritable carotid sinus in his neck, and fell dead. There is no method for confirming carotid sinus reflex or disproving it as a cause of syncope and death.

Case 7.—A case of hypersensitivity, as death after the administration of a few drops of a dilute sodium thiopental (Pentothal) solution or of cocaine, will not yield a demonstrable mechanism of death at autopsy.

Case 8.—When a patient dies as the anesthetic mask is being applied, before any anesthesia or other operative procedure is initiated, no cause of death may be found, even after thorough investigation by any present methods.

Morphologic Lesion.—In most deaths where gross autopsy findings in the form of acceptable morphologic lesions are at hand, the immediate cause of death is functional or physiological. In many cases there may be no certain association between the morphologic lesion accepted as the cause and the actual final mechanism of death.

This may occur in coronary cases, except in the instance of ruptured myocardial infarct with tamponade. While morphologic lesions of the coronary vessels may often indicate coronary insufficiency, as long as such changes were present for a considerable time before death the visible morphologic explanation does not really account for the death. Often there is no mention in the history of a sudden exertion that would permit acceptance of a sudden relative insufficiency. Very extensive disease of the coronary vessels may not occasion symptoms prior to death. At many autopsies, patients are found with extensive coronary disease; yet this finding is incidental to an independent extraneous mechanism as the cause of death. The presence of a gross infarct precludes the initial coronary occlusion giving rise to it as the only or direct cause of sudden death. This is true for such occlusion by thrombus, embolus, hemorrhage into atheroma, the dissecting elevation of a value-like intimal flap, or occlusive discharge into the lumen of grumous atheromatous material, if infarct changes of some duration are present. A fresh extension of the thrombus is not a common terminal mechanism. Occasional observation of gross myocardial infarct without coronary occlusion also suggests that physiological mechanisms can be very significant. Healed extensive myocardial infarcts so frequently found at autopsy also make inadequate the mere presence of gross infarction as an infallible cause of death. All other lethal mechanisms must be excluded to give such a lesion its full significance in evaluating the cause of death.

Physiological and Pathological Mechanisms.—Another common instance in which pathological findings do not explain the physiological mechanisms of death is in death after lobar pneumonia. Impairment of function of the involved lung does

not usually account for the death; actually, it is the toxic effect on the heart and the viscera, on the general circulation and the peripheral vessels, and on the central nervous system and specialized centers that has greater weight. While attention at autopsy may be focused on the lung lesion, the more significant mechanism of death may be a subsequent complicating finding of meningitis or bacterial endocarditis. Many patients recover from the same extent and type of pneumonia when such complications do not ensue.

Physiological mechanisms often kill with no demonstrable positive findings, and morphologic lesions are significant only to the extent that they produce lethal physiological distortion. Only when all other mechanisms of death can be excluded are morphologic lesions per se, without demonstrable lethal physiological mechanisms, valid as the cause of death. It is essential that the autopsy be complete and that the pathology found be incompatible with life in order to be acceptable as a cause of death. The familiar examples cited show that the terminal undemonstrable physiological component in the mechanism of death can be separate and independent of the demonstrable dramatic positive findings.

Death may be defined as the cessation of integrated life functions. Life depends on integration of the following physiological functions: ingestion, digestion, absorption, respiration, distribution (circulation), integration (nervous system and endocrines), metabolism, excretion, and egestion (elimination). Death occurs if any of these functions is much impaired or arrested. A long period of symptoms usually precedes death when impairment occurs in the three first and three last listed functions.

Medicolegal cases are concerned with instantaneous, sudden, and unexpected deaths. Disturbances in respiration, circulation, and the central nervous system result in deaths by asphyxia, syncope, and coma. Involved in each is the failure of oxygen to reach the tissues.[8] Moritz [1] found that vascular accident was the most frequent cause of sudden death.

Degree of Certainty.—To overcome the confusion in evaluating causes of deaths, let us classify the main findings in the interests of exact degree of certainty as to the mechanisms involved.

First, a finding occurring during life may be incompatible with life. The finding may be morphologic (gross or histological), bacteriological, or chemical. This offers the only certain cause of death. For instance, a ruptured heart may be found at autopsy, with a massive accumulation of blood in the pericardium, or a heart may be found free in the thoracic cavity as a result of traumatic compression of the chest. There may be a lethal quantity of cyanide in the brain or a lethal quantity of carbon monoxide in the blood.

Second, a positive finding adequate to cause death may also be known to be compatible with life. After excluding other possible mechanisms of death, it is reasonable to infer that the positive finding was the cause; some physiological mechanism in addition to the morphologic distortion is inferred and accepted. This constitutes the largest group. For instance, a compound fracture of the tibia and pelvis may be caused by an automobile accident, and the patient may die with symptoms of shock. Further, the ordinary case of marked coronary sclerosis may be seen, with a few areas of hemorrhage deep in the papillary muscle suggesting insufficiency. In this category also is extensive lobar pneumonia.

Third, there may be no findings known to cause death. In this group, the cause often remains undemonstrable, so that in immediate and sudden deaths circumstantial data may be of considerable importance. Physiological mechanisms may apply here exclusively. In my experience, the cause of instantaneous death is often not found at gross autopsy. Deaths caused by acidosis, alkalosis, disturbed potassium metabolism, vagal reflexes, and sudden cardiac decompensation leave no morphologic expression.[9] In this group are the hypoglycemic reaction after overdosage of insulin and the death that follows "pleural shock" as the result of the introduction of a needle for a pneumothorax.

This grouping would help an attorney or jurist to evaluate probability of error and to decide the validity of the stated cause of death, particularly in the second and third categories, where a basis for the opinion of the cause of death should be established.

When multiple natural causes or both natural and unnatural causes, such as trauma, are involved, classification becomes more complex. The timing of the separate entities is often important. Natural causes arise from within the body, unnatural usually from an external source. The following examples illustrate this: 1. Trauma alone causing death: A body is mangled by a railroad train. 2. Natural disease alone causing death: A ruptured dissecting aneurysm occurs with cardiac tamponade. 3. Trauma as a minor contributory cause of death: A patient admitted to hospital with fractured elbow dies there of uremia secondary to hypertrophied prostate and ascending infection. 4. Natural disease as a minor contributory cause of death: A ruptured liver with luetic aortitis and ostial stenosis causes death. 5. Natural disease and trauma both significantly involved in the cause of death: A man develops a coronary thrombus and falls from a scaffold, with lesions of considerable extent in both categories. A hypertensive patient with history of a previous stroke falls down stairs and shows a fractured skull with an intracerebral hemorrhage. A ruptured spleen follows minimum trauma in a patient with chronic myelogenous leukemia.

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Careful analysis of the unnatural causal component interrelations is required in medicolegal cases, for justice is best served if the sequence and interrelationship of all facts is established. 1. Trauma directly causing natural disease: A man is injured at work and develops local infection and then generalized sepsis. This would include, too, a man who is struck on the leg and, after an interval, develops an osteogenic sarcoma. 2. Trauma indirectly causing natural disease: An aged male fractures his femur as a result of trauma and develops bronchopneumonia. Included here also is the case of extensive multiple injuries which lead to shock, a fall in blood pressure, and a secondary coronary thrombosis. 3. Trauma aggravating preexisting natural disease: A patient with slowly growing tumor of long duration shows widespread dissemination after trauma. An instance of melanoma or teratoma of testis is an example. 4. Natural disease causing trauma: A patient develops fleeting attacks of blindness because of brain tumor and is injured in an automobile accident during an attack. 5. Trauma and natural disease unrelated (the natural disease may be preexisting or coincidental): A breast tumor may be struck and the increased growth is found to be due to fat necrosis. Included also is a patient who dies of profound anemia with hemorrhage from ulcer of the stomach, and lead poisoning contributes to mechanism of anemia.

In the relationship of natural and unnatural, such as trauma and tumor, the time factor is most significant.[6] What has been said for trauma applies also to other conditions of unnatural cause, such as poisoning, asphyxia, and drowning. The analytical approach can be used to establish the part played by the unnatural component to determine the medicolegal significance in criminal and civil actions.

Problem of Definition

"Cause of death" allows for various definitions and interpretations. Does the request for cause of death call only for the actual terminal mechanism, with basic disease considered contributory? Different death certificates in use in the United States request "immediate or determining cause of death," "immediate proximal cause of death," "primary cause of death," and "disease causing death." Clear definitions are needed of "main disease," "basic disease," "contributory cause of death," and "complicating cause of death" (table 1).

The United States Census Bureau, to avoid ambiguity, requests the "immediate" cause of death and the "underlying" cause of death. The first is defined as the last of a series of disease entities which contribute to death. The latter is defined as the disease entity which initiates the series of disease entities resulting in death.

In certifying a death, the physician's answers are now limited to terminology acceptable to public health officials. His failure to adhere to specific diagnostic terms as listed in the Manual of International Statistical Classification of Diseases, Injuries, and Causes of Death (Sixth Revision, World Health Organization, Geneva, Switzerland, 1948) or the Joint Cause Manual and any attempt on the part of the physician to qualify his diagnosis can cause difficulties and delays for the family, the undertaker, and himself.

The prevailing concept of single underlying cause introduces trying and at times insurmountable difficulties and requires arbitrary decisions by the certifying physician or the coding process. In chronic illness, often multiple, related, and, at times, unrelated heterogenous causes prevail. The expansion of the "disease-complex" or "associated cause" classification would be more applicable. Yet, mere expansion of the present Vital Statistics Instruction Manual cannot substitute for the individual judgment of the certifying physician personally acquainted with the specific facts and complex ramifications of the case.

Recommendations

By the present procedure for obtaining medical information on the death certificate, all objectives are merged in the listing of the cause of death. In my opinion, a separate statement assuring that no crime was committed and that death was due to natural causes should be required. Then a separate parallel listing should be made of the sequence of the medical events preceding death and another separate listing of the part each pathological or clinical entity played in shortening the life of the patient. Such listings are often not similar. If not identical, sequence and significance cannot then be listed in a single order simultaneously. For the first listing of sequence, replies in proper order should be required to the question, "Which was the initiating disease entity, and what was the relationship in the order of their development of the subsequent complications or the simultaneous existing entities?" To determine the significance of each entity in causing the death, so important for statistical purposes, the second separate listing should call for an answer in the order of weight to the question, "What are the disease entities or complications in order of magnitude, i. e., without which death would have been delayed the longest or life prolonged the most?"

A true meaning can thus be given to "cause of death" by the separation of the objectives in certification. It is essential to make the concept of pathogenesis and sequence distinct from the concept of significance and actual contribution to the mechanism of death. The physician is helped by thus delimiting the definition of the phrase "cause of death," and he can offer the exact information required and avoid confusion and error. The form

of the certificate on "page 2" of table 1 promotes this endeavor. Table 2 is an illustrative example of the proposed medical report.

The average physician who is not a pathologist certifies relatively few deaths in his lifetime; he can hardly be expected to become expert in the field of certification. Capable physicians are often profoundly ignorant of the issues and nuances involved —bear witness the frequent instances of obvious preferred form of listing of the diagnoses with a uniformity which does not now prevail. The "removal and embalming permit number" for the undertaker could be obtained at this time by the physician and entered on the certificate to expedite the removal of remains.

It would then be unnecessary to divulge the cause of death to the mortician when the confidential certificate is used. The sealed confidential certificate for the cause of death could then be used with greater assurance of success. On this score, it should be noted that during the very first year of use of the sealed confidential certificate in Manhattan (where no one but the physician and the certification clerk ever saw the certified cause of death), there was a 10% drop in recording of arteriosclerotic heart disease and a corresponding increase in recording of alcoholism, syphilis, cancer, and insanity on death certificates.

TABLE 1.—*Certificate of Death by Natural Causes and Proposed Medical Report*

Page 1

Name..... Social Security No. ..... Certificate No. .....

Personal Data

Residence .....
.....
Social status .....
Date of birth .....
Age .....
Wife or husband .....
Occupation .....
Burial place .....
Citizenship .....
Veteran of war of .....
Father's name .....
Father's birthplace .....
Mother's birthplace .....
Informant's relationship .....
Signature of funeral director .....
Burial permit number .....
Place of burial .....
(Completed by mortician)

Administrative Data

Place of Death .....
.....
Length of stay at place of death .....
Date and hour of death .....
Sex .....
Color or race .....
Approximate age .....

I hereby certify that (I attended the deceased)
(A staff physician attended the deceased)
from ..... to ....., and that the death ..... (was) .....
(date) (date)
..... (was not) ..... caused directly or indirectly by accident, homicide, suicide, acute or chronic poisoning, by abortion, or suddenly while in apparent health, without care or attendance by a physician, or in any suspicious or unusual manner.

.....
Signature

Unless this statement can be signed, the case should be referred to the medical examiner or coroner.
I further certify that the death ..... (was) ..... (was not) ..... due to communicable disease listed in the Sanitary Code which required that the casket must be permanently sealed before removal from the place of death.

.....
Signature
(Completed by physician)
.....
Address
Removal permit no. .....
.....
Telephone number

Page 2

Name..... Place of Death..... Date of Death.....

Factual Data	Cause of Death (Pathogenesis)	Cause of Death (Significant)
Outstanding and pertinent facts of present illness (derived from symptoms, other facts of history, physical signs, laboratory data, or other means which serve to aid in arriving at a diagnosis). Autopsy findings. Degree of Certainty*	List and group diagnosis of present illness in sequence of occurrence (from standpoint of initiating entities and complications).	List diagnoses in order of significance and contribution to the cessation of life, i. e., in the order of severity, or lesions without which life might have been prolonged the longest.
The cause of death is based on (check headings or delete captions which do not apply).		
Past history..... Direct clinical observation.....		(Substitute a running and analytic narrative only if preferred)
Present clinical findings only.....		
Hospital study.....		
Was there consensus of opinion.....		
Operation with or without biopsy and histological study.....		
Laboratory tests, as isolation of specific etiological agent (bacterial or viral)..... recognized serological procedure..... biochemical studies..... toxicology..... other tests.....	List other independent pathological conditions contributing to mechanism of death.	
Autopsy without..... or with..... confirmatory histological studies.		
Evaluate statistical significance of this case by writing whether this diagnosis has been established with sufficient certainty to warrant inclusion in analytical studies. Yes..... No.....		
Should this case be investigated from standpoint of contacts? Yes..... No.....		..... Signature

* May or may not appear on final blank.

medicolegal cases certified by a physician in good faith on a natural death form when clearly the cases should have been referred immediately to the coroner or medical examiner. In modern society, with a telephone always at hand, a discussion or consultation should be held with an expert in certification prior to completion of the certificate. This routine would clarify jurisdiction at the outset and thus obtain agreement on satisfactory terminology in keeping with acceptable nomenclature and the

The sealed form had to be abandoned in New York City, because without prior consultation many certificates had to be rejected when finally presented for filing by the undertaker for burial permission just prior to the funeral. The sealed cause of death often showed trauma in the listing and other diagnoses with legal implications which varied from homicide to poisoning. The resulting last-minute delays in scheduled funerals made it essential to vary the procedure and to divulge the cause of death to the undertaker on an open form. He, in turn, then calls the certification clerk for his removal and embalming number, checking the propriety of the certification. This is archaic, for if there is any question about the diagnosis, the undertaker is hardly in a position to resolve it. In this way at least, if the diagnosis is found erroneous, the case can be referred to the legal authorities with less delay and before the body is embalmed.

Summary

Certain limitations in administrative procedure in certification exist along with inadequacies in the present method of determining the cause of death. The interplay of forces in the cause of death often defies analysis or makes it most difficult. Therefore, consultation prior to completion of the certificate with an expert in certification or responsible authority is recommended, as is a revision of administrative procedure by separating the sequential from the significant to avoid ambiguity in gathering and recording data of the individual clinical and pathological entities.

The application of a physiological classification of causes of death and the documentation of the source of the facts and the basis for the diagnosis, to determine the "degree of certainty," is also advisable. An evaluation of accuracy would follow.

TABLE 2.—*Illustrative Medical Report*

Name..... Place of Death..... Date of Death.....

Factual Data	Cause of Death (Pathogenesis)	Cause of Death (Significant)
Outstanding and pertinent facts of present illness.	Grouped listing of diagnoses of present illness.	Diagnoses listed in order of significance in contributing to death.
A 65-year-old male develops urinary obstruction which requires relief by suprapubic cystotomy, and two weeks later a suprapubic prostatectomy is done. Five days after this operation, the patient suddenly gasps, becomes cyanotic, and dies within the hour with a diagnosis of pulmonary embolism. Autopsy showed no pulmonary embolus but a definite coronary thrombus.	Coronary thrombosis, left anterior descending Prostatic hypertrophy with urinary retention Cystotomy Suprapubic prostatectomy Pyelonephritis, recent	Coronary thrombosis Acute pyelonephritis
Degree of certainty*	Incidental and independent pathological conditions	
Diagnoses made on what basis? Autopsy with histological studies (check)	General arteriosclerosis Nephrosclerosis Old healed apical tuberculosis	
		..... Signature

* May or may not appear on final blank.

By prior consultation, the physician would be spared the embarrassment and irritation that follows such an error when the certificate is necessarily rejected by the certification clerk and the body is belatedly referred to the displeased medical examiner or coroner. At best, a new certificate may be required of the physician, with all that this implies in loss of time and temper.

The physician is often unaware of the implication of his opinion expressed on the "open" certificate when it is subsequently used as a legal instrument. As long as the certificate is admitted in evidence, provision should be made for evaluation of its accuracy. Some recommendations on this score have been made elsewhere.[7] The basis for his opinion should be made available. Difficult and controversial problems in certification could be reviewed by a board of pathologists or an impartial panel similar to what has been used in negligence litigation in New York County so successfully for the evaluation of medical facts and opinions.[8]

Above all, the accuracy of vital statistics would be enhanced. To achieve these objectives, the changes in certification procedure outlined above are suggested and the incorporation of improvements in the death certificate proper recommended. A board of pathologists or an impartial panel of experts to examine death certificates in difficult or controversial cases would increase accuracy.

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PRINCIPLES FOR PRECAUTIONARY LABELING OF HAZARDOUS CHEMICALS

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A substantial number of packaged chemicals are not now required by law to identify hazardous ingredients on their labels or to warn users of the dangers of overuse or misuse. The need for precautionary labeling of chemical products was first formally registered by the American Medical Association in 1884 with the adoption of a resolution urging that Congress and the several state legislatures be called on to enact legislation to require lye to be sold as a poison and under a poison label. A broad and encompassing act requiring precautionary labeling of hazardous substances in commercial, household, and industrial chemicals has now been drafted by the American Medical Association's Committee on Toxicology. The bill is intended as a model for uniform laws to require the declaration of hazardous ingredients and warning statements on the label and in the accompanying literature of chemical products.

The accelerating pace of modern technology is most evident in the multitude of chemical products which are used in industry, on the farm, and about the home and its premises. A quarter of a million or more trade-name substances are available, and their number and variety increase with each successive year. For example, an estimated 70,000 preparations are or have been marketed as cosmetics, a comparatively small class of chemical products. It has been further calculated that as high as 1,000-2,000 new cosmetic products appear each month, many of which disappear shortly thereafter.[1]

A substantial number of packaged chemicals are not now required by law to identify hazardous ingredients on their labels or to warn users of the dangers of overuse or misuse. A recent survey revealed the sketchy, nonuniform, and generally inadequate pattern of regulations at the state and national levels for informative labeling of chemical products.[2] The survey disclosed that, at the national level, label laws exist for drugs, pesticides, certain caustic and corrosive substances, and narcotics. There are no federal laws for the precautionary labeling of industrial chemicals, household chemicals, or poisons, although regulations for the packaging and labeling of dangerous chemicals and explosives in transit by land or water or via the mails are issued by the Interstate Commerce Commission and the Post Office Department.

State legislation is a patchwork of laws patterned after *(a)* existing federal legislation, *(b)* earlier but now superseded federal statutes, and *(c)* special laws which are often overlapping and sometimes even contradictory. Various types of special laws for chemicals are in force in the states, some of which have as many as five such statutes. On the other hand, other states have inadequate or no regulations.

The Need for Precautionary Labeling

The need for precautionary labeling of chemical products was first formally registered by the American Medical Association in 1884 with the adoption of a resolution urging that Congress and the several state legislatures be called on to enact legislation to require lye to be sold as a poison and under a poison label. Later resolutions were adopted in 1910 and 1918 urging precautionary labeling of caustic poisons. Through the untiring efforts of Drs. Chevalier Jackson, Charles W. Richardson, and William C. Woodward, a Federal Caustic Poison Act was enacted in 1927.[3] State laws uniform with or generally parallel to the federal law have since been enacted in 25 states.

Despite these early expressions of organized sentiment for the labeling of articles with poisonous ingredients, many chemical products have remained essentially free of federal or state labeling regulations. The first state to move into this vacuum was California, which formulated a code for hazardous chemicals used in industry in 1944. Several states have since adopted similar standards. The first state and municipality to modify and extend these standards to embrace household chemicals were New York state and the city of New York, which simultaneously developed amendments to their sanitary codes in 1954. Since then a smattering of states (Connecticut, Indiana, Kansas, Massachusetts, North Carolina, and Texas) have adopted widely varying regulations intended to require informative labeling of poisonous materials.

The compelling need for fully informative labeling of chemicals has been reflected in correspondence received by the American Medical Association after it made known its interest in this problem. The following excerpts from recent correspondence indicate the types of complaints which are prevalent and the general concern for appropriate legislation.

Secretary, Committee on Toxicology, American Medical Association.

A university professor writes:

My 3-year-old child ingested the dye in question accidentally. It had been kept in a relatively safe place but due to the fact that it was not labeled "poison" or dangerous, no unusual precautions had been taken.

The child in question is one of five in the household, ranging from 6 months to 7 years, and this is the first instance of such an accident. This, I think, speaks for itself as to the amount of precaution taken in the household.

It may interest you to know that I contacted the company that manufactures the drug in search of information and can truthfully say that their cooperation was far from pleasant or effective.

I wish to thank you for the information which you provided me and hope, among other activities of the A. M. A. that a recommendation for legislation for simple measures to obviate such accidents in households will be considered.

A dermatologist writes:

I want to bring to your attention a very important matter as it affects the practicing dermatologist. I recently saw a garage worker, who does body repair work primarily, for a severe dermatitis affecting the hands and forearms, face, neck, and upper torso. He was unable to perform any work. As he improved, a series of patch tests were performed. Contact tests were done with all materials suspected of having solvent in them. A contact test was performed with . . . . and a drop of this on his skin produced a flare of erythema. Twenty-four hours later there was a papulovesicular rash at the site which exactly reproduced the clinical dermatitis.

Since this man earns his livelihood in the body repair business, he was most anxious to know the nature of the substance in the remover that was causing his trouble. Naturally, he had a horror of a recurrence. I wrote to the manufacturer of the metal cleaner, and enclosed is a copy of the reply. In fact this type of reply is so common that I do not write unless the patient requests it. For the most part, where one writes inquiring for the composition of cosmetics that have produced a dermatitis, and many other different kinds of products, as a rule we get a brush-off. Yet, I think for the individual mentioned above, it is vitally important for his own future health that he know the nature of this chemical. It might be the hydrocarbons in there, and, in turn, it could be something else. If he knew and his family doctor knew, future trouble might be prevented.

I certainly wish your Committee success, and hope it will bring about a uniform labeling law.

In response to the dermatologist's request, the firm replied as follows:

With reference to your letter of January 21, 1957, the product you cite is one that is being used by literally thousands of concerns, with no ill-effects such as you mention.

However, our medical advisor states that when exposure to a product containing hydrocarbons does result in a condition such as described in your letter, the best means for relief is the discontinuance of such exposure, and that the authenticity of this opinion is confirmed by its precept being widely followed.

Therefore, any need for furnishing formula, samples, or information, other than that our product is a combination of ingredients of which some are hydrocarbons commonly and widely used for the purpose indicated by the name of the product is, in our opinion, dissipated by the above.

The foregoing reports are not representative of the attitude of the major chemical industry trade associations who have or are developing programs of precautionary labeling for their membership. Nevertheless, continuing complaints of this nature which are reported suggest that they are representative of the attitude of the less conscientious purveyors of these products. The experiences described in the above letters refute oft-repeated objections to precautionary labeling; namely, that trade secrets would be violated, that warning statements are a handicap, or that precautionary labeling is useless since small children cannot read labels. The fear that trade secrets are lost through label declaration of hazardous ingredients is ill-founded, and happily fewer firms now have this attitude. Experience has also demonstrated that warnings against misuse do not frighten away customers, rather they engender confidence that the product can be safely used if directions are followed. The claim that such labels are useless because the most frequent victims of poisoning are small children who cannot read the warnings is a spurious argument. Child poisoning occurs because harmful substances are easily accessible, less because of overt carelessness on the part of the parents than of their failure to realize that many familiar products are capable of harm. Label warnings are intended to counteract this easy familiarity which breeds carelessness in the handling and storage of chemical products.

Improvement is also needed in the labeling of those household and commercial chemical products which now carry some type of precautionary labeling. This was recently demonstrated at an industrial safety congress where a random sample of household and commercial chemical products were purchased for display.[4] Examination of a widely sold dry cleaner revealed that it was labeled in large letters "SAFE TO USE" "cannot be burned or ignited unless it is heated to beyond 110°F" and "No Dangerous Fumes." However, tests on the product disclosed that it possessed substantial inhalation and flammability hazards. About 15 other products were similarly examined, and all of them were found to be wanting with regard to adequate or accurate label warnings.

Development of a General Labeling Law

Legislation to require precautionary labeling of all hazardous chemical products has been under consideration by the American Medical Association for a number of years. In 1926, a proposal for a general federal law to safeguard the distribution of all poisonous substances in interstate and foreign commerce was proposed. This proposal was apparently set aside to assure passage of a labeling statute for caustic and corrosive substances. More recently, in 1951, a resolution of the House of Delegates called for a general study of the problem. The Reference Committee on Hygiene and Public Health strongly endorsed this resolution and stated that the "Board of Trustees plans to take appropriate steps to warn the public and parents of young children of the dangerous nature of household products known to be poisonous but not so labeled. . . ."

The Committee on Pesticides was called upon to implement this resolution. During the next two years it compiled information and explored possibilities for increasing public understanding of the uses and limitations of household chemicals. For educational purposes, the Committee prepared a medical exhibit on accidental poisoning and a pamphlet depicting typical poisoning cases and sponsored a radio transcription "Defense Against Poisons" for use by local medical societies. These activities culminated in an exploratory meeting in January, 1954, a general conference in September, 1954, on the health problems of household chemicals which led to the formation of the Committee on Toxicology in January, 1955. In October, 1956, as a result of prior conferences with industry, the Board of Trustees authorized this Committee to draft a model law for the labeling of dangerous household and industrial chemicals.

In considering the provisions of a model law for labeling of all types of hazardous substances, the Committee has adopted a set of principles which are fair and reasonable and reflect current knowledge of the toxicity of chemicals and the conditions under which they are employed.

The principles are as follows:

Coverage.—Precautionary labeling should be applicable to all chemical products containing hazardous substances which are not now so specifically regulated. These include packaged chemicals intended for home, commercial, or industrial use but excluding substances already informatively labeled and subject to existing statutes such as foods and drugs (Federal Food, Drug, and Cosmetic Act of 1938), economic poisons (Federal Insecticide, Fungicide, and Rodenticide Act of 1947), the caustic and corrosive substances in the concentrations specified under the Federal Caustic Poisons Act of 1927, and state laws in basic agreement with these statutes. In effect, the Committee's recommendations are intended to supplement rather than supplant existing legislation wherever applicable and practical.

The same labeling standards should apply to chemicals for export to foreign countries as to those for domestic consumption. This would obviate the recurring complaint that less than standard products are sold to foreign purchasers. Many countries, especially those without specific legal requirements, allow merchandise to be accepted within their borders on the basis that it complies with American law. Consumers of exported chemicals are entitled to the same protection that domestic users receive. Therefore, exemptions in labeling laws which allow a double standard for chemicals in domestic and foreign commerce should be scrupulously avoided.

Toxicity Standards.—The lack of adequate standards is a common failure of proposed legislation. While exact standards cannot be established when accepted and applicable testing methods are lacking, regulations should be constructed to allow those reading the law to determine the extent to which it may apply to their products.

The temptation to base toxicity standards on undefined human experience should be avoided when no measurement of this is provided. However, the toxic hazard of a substance or mixture of substances can be based on animal tests, since they provide a consistent and usually reliable index of the poisonous properties of a chemical. For the purposes of legislation, a toxic (poisonous) substance is tentatively defined as any material which kills half or more of a group of laboratory animals (rats) within two weeks in a single oral dose of 5 Gm. per kilogram of body weight or less, or on skin contact for a period up to 24 hours in a concentration of 1 Gm. per kilogram or less, or on inhalation for one hour or less in a concentration up to 200 mcg. per liter of gas, vapor, mist, or dust.

In the determination of acute toxicity, mortality data should not be computed on observation periods of less than one week and preferably over a two-week period, because of the possibility of serious errors occurring in the interpretation of the true inherent toxicity of compounds. Even within such a closely related series of compounds as the sulfanilamide drugs, significant differences in survival times after lethal doses are exhibited. This is due to differences in solubility, the least soluble possessing the greatest latent toxicity. Latent toxicity may not manifest itself within the usual 48-hour observation period, therefore a much greater time interval is needed for its detection.

The term "poison" could be abandoned in view of the wide variation in legal and scientific definitions of the term which now exist. Most, if not all, of the legal definitions are based on criteria which many now consider to be outdated. Existing statutes either specify values varying from 5 grains (0.3 Gm.) to 5 Gm. as the lethal quantity for (adult) human life or they resort to indefinite terms such as "noxious" or "virulent poison." Those statutes that confine the term to highly toxic compounds (LD_{50}—50 mg. per kilogram or less) fail to include such common causes of fatal accidental poisoning as carbon tetrachloride and kerosene. Nevertheless these are dangerous chemicals, especially to the very young who may die from as little as a mouthful (15 cc.) of kerosene. Finally, the possibility of proposed or pending litigation over the issue of the poisonous character of certain types of drug products may jeopardize the status of some legal definitions of this term.

In 1916, the United States Public Health Service, in a review of laws and regulations relating to poisons and habit-forming drugs, found that a lack of uniformity in various state laws and a lack of a standard definition of poison were the two most

serious defects at that time.[4] State narcotic laws uniform with the federal Harrison Narcotic Act have since been enacted in the various states. Regrettably little improvement has become evident in the definition of poison over the last 40 years. Therefore, in lieu of a generally accepted definition of poison, substances capable of causing death to any age group are dangerous chemicals and should be so designated on the label.

Strongly sensitizing chemicals, which have heretofore not been considered in general labeling legislation, should be required to be identified and carry warning statements on their labels. The frequency and severity of the sensitization reaction determines whether a sensitizing material offers a significant potentiality for harm. An extensive dermatitis or other type of moderately severe disability occurring at a frequency of 1:10,000 would fall within the meaning of this definition.

Legal precedent for the labeling of strong sensitizing chemicals may be found in the special labeling requirements for paraphenylenediamine, an ingredient of certain hair dye preparations. While the majority of legal decisions suggest that the manufacturer of a product has no legal responsibility in either warranty or tort to the allergic plaintiff, legislation to recognize the allergic or unusually susceptible as a legally identifiable class is long overdue.

A survey of 71 allergists and dermatologists constituting approximately 1% of the membership of the American College of Allergists and the American Academy of Dermatology and Syphilology disclosed almost unanimous support for including and defining this group of chemicals as a separate category of hazardous substances to which precautionary labeling should be applied.

Labeling.—The labeling of hazardous substances should be based on the anticipated degree of danger. Under various suggested labeling schemes several types of hazards (such as toxicity and flammability) are recognized. These are identified by a short statement of the hazard and by appropriate signal words such as "poison," "danger," "warning," and "caution" to signify progressively lessening degrees of hazard. In practice, however, few persons are sufficiently interested or informed to analyze the precise shades of meaning given to the signal words and thereby act accordingly.

Many users of hazardous substances are not likely to draw a practical distinction between "danger" and "poison" and even fewer are likely to appreciate the difference between "warning" and "caution" on the label. However, the use of familiar symbols such as a flame for inflammable substances or a skull for a poisonous substance are readily recognized and meaningful even to the illiterate. The use of carefully chosen symbols in conjunction with the signal word "danger" for substances presenting the greatest hazard would facilitate recognition and bridge the language barrier.

Labeling for this group of products should include not only the label itself but other printed and graphic matter accompanying the product. This permits some control of advertising, especially as it relates to claims for safety. The re-use of food, drug, or cosmetic containers still bearing original labels or identifiable as such by characteristic shape, impression, or closure as containers for hazardous substances should be prohibited.

Conclusions

Precautionary labeling of hazardous chemicals should not be considered as an easy panacea to the difficult problem of poisoning prevention. This problem is far too complex to be solved by such a simple procedure. Nevertheless warning labels and the declaration of hazardous ingredients form the keystone on which to base social remedies for increasing public awareness of hazards and encouraging careful handling and storage of the many possibly harmful chemical products now available.

To meet this need, a broad and encompassing act requiring precautionary labeling of hazardous substances in commercial, household, and industrial chemicals has been drafted by the American Medical Association's Committee on Toxicology. The bill is intended as a model for uniform laws to require the declaration of hazardous ingredients and warning statements on the label and in the accompanying literature of chemical products. Widespread interest has been manifested in this measure, as evidenced by the many requests for copies of the bill and the opinions given on the means for initiating legislation. At the present time, such interest has been registered by state and federal legislatures, state and national medical societies and allied health groups, state and local health departments, and the Council on State Governments. Distribution of the bill is presently being made. Notices of intention to hold conferences for discussion and emendation of various legislative proposals are being circulated to interested parties in government, medicine, and industry.

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CLINICAL NOTES

THROMBOPHLEBITIS OF SUPERFICIAL ABDOMINAL VEINS

AN UNUSUAL COMPLICATION OF INFERIOR VENA CAVA LIGATION

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and
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Thrombophlebitis of the superficial abdominal veins is not serious nor a potenially dangerous complication of inferior vena cava ligation; however, we have been unable to find a similar case report in a review of the recent English literature. We believe that this case may be related to the superficial thrombophlebitis of the breast and chest wall [1] often reported as Mondor's disease, although, in this patient, a lower abdominal superficial thrombophlebitis was present. Furthermore, there is no mention of prior inferior vena cava ligation in the reported cases related to so-called Mondor's disease.

The usual complications of inferior vena cava ligation are of such protean nature as to be of no particular interest with regard to the individual case report. It is worthy of note that some [2] report a significant lack of postoperative complications, whereas others [3] indicate many difficulties. The procedure in their opinion should be done only as a lifesaving measure.

This obese patient had a ligation of his inferior vena cava after multiple pulmonary infarcts from an undetermined site of origin. He had leg edema postoperatively and was treated with bed rest for two weeks and elevation of his lower extremities, which were wrapped with elastic bandages. Anticoagulants were not given. About eight months postoperatively he developed phlebothrombosis of a single vein in the left lower quadrant of his abdomen extending up to the costal margin anterolaterally. This was followed in about two weeks by an acute inflammatory change (thrombophlebitis) in the same vein and its adjacent tributaries. Shortly after hospitalization it became evident that he was wearing his belt and pajama string tightly bound around the costal margin at the exact site at which the thrombophlebitic process began. This in association with his obesity predisposed toward venous stasis in compensatorily dilated veins.

Report of a Case

A 58-year-old male carpenter was injured on Sept. 27, 1954, at which time a power saw lacerated his right thigh just above the patella. This injury was adequately handled with minor surgical repair, and no particular difficulty arose until two weeks later when, while still ambulant, he suddenly began to have pain in the left side of his chest. This pain was very severe, was pleuritic in character, and required narcotics for relief. The electrocardiogram was within normal limits. After a few days, he felt well and remained that way until about two weeks later (four weeks after his injury to the thigh), when he noted a second onset of this pleuritic type pain in the right side of his chest. Several days later, he began having hemoptysis.

Physical findings observed after his second onset of pleuritic pain included diminished breath sounds with rales over the right lower posterior part of the chest. No murmurs were heard and the heart was not enlarged. The liver and spleen were not palpable, and there were no peripheral signs nor any indication of thrombophlebitis or phlebothrombosis. X-ray of the chest showed some "pneumonitis" at the base of the right lung with obliteration of the left costophrenic angle. Electrocardiograms at this time revealed abnormalities strongly suggestive of a diagnosis of instrinsic heart disease, but it was felt that they could also be associated with pulmonary infarction. The source of possible thromboembolism was not clear, but it was felt that any part of the venous system or of the chambers of the right side of the heart was of course, a potential area of origin. Subsequent chest x-ray revealed the left costophrenic angle to have cleared up, but he had developed a definite increase in his heart size and the right diaphragm was noted to have increased in height, with an area of clouding noted to be present in the right costophrenic angle.

The patient suffered two further attacks of chest pain which necessitated ligation of the inferior vena cava on Nov. 2, 1954 (performed by Dr. Alfred B. Longacre), about five weeks after the minor trauma to the right thigh. The electrocardiograms then showed regressive changes of the previously noted abnormalities, and it was thought that the site of origin for the thromboembolism was the venous system peripherally rather than in the heart itself.

The patient was treated with bed rest, elastic bandages, and brief periods of ambulation after about two weeks and he was not given anticoagulants postoperatively. He did fairly well over a period of months, with the exception of development of extreme edema of his two lower extremities. He had no specific trouble at any time with his right lower extremity. Postoperatively he maintained edema of both lower extremities—greater on the right (side of the minor laceration).

In early July, 1955, he noted a slow development of a cord-like structure in the left lower quadrant of his abdomen, which he likened to a "pencil mark" extending from the region of the costal margin downward to Poupart's ligament. This progressed in size to a total of 9 in. in length and assumed grayish-green or brownish discoloration. There was a

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palpable cord about 0.25 in. in diameter, with the discoloration about 0.5 in. in breadth. Under observation, this cord-like structure changed, becoming involved in an inflammatory reaction in association with three of its tributaries in the same general vicinity. This then was associated with swelling, redness, and increased local heat. In addition, he had a low-grade temperature elevation for a short while. Heart and lungs were normal and blood pressure was 140/90 mm. Hg. The electrocardiogram and chest x-ray were normal. The general physical examination revealed no abnormalities other than the presence of moderate pitting pretibial edema bilaterally (slightly more on the right side), unrelated left prepatellar bursitis (which was later interpreted as possibly being due to a gouty diathesis), and prostatic fluid loaded with pus cells. The patient gave a history of having been treated previously for chronic prostatitis. The sedimentation rate was elevated. Special blood studies by a hematologist revealed no evidence of blood dyscrasia. There was no evidence of any deeper abdominal symptomatology nor disease, and, therefore, the possibility of pancreatic pathology as a possible diagnosis was discarded.

The patient was treated with absolute bed rest, anticoagulant therapy, multivitamins, prostatic massage, sulfisoxazole (Gantrisin) tablets, and general supportive measures while in the hospital for a total of 23 days. During this time, he wore elastic stockings up to the knees, including the toes and heels. He was placed on a weight-reduction diet at this time, with his original weight being 208 lb. (93.4 kg.).

The acute thrombophlebitic process subsided rapidly and he was discharged to his home on therapy with anticoagulants and vitamins in gradually diminishing doses. Prostatic massage and sulfisoxazole therapy were stopped while he was still in the hospital. He was given a few doses of crystalline trypsin (Parenzyme) (as recommended by Bowers[4]) intramuscularly for its possible effect on decreasing the thrombophlebitic process. Within several weeks after his discharge from the hospital, and with the utilization of the elastic stockings, the patient lost most of the edema of his legs and was seen on multiple visits in the office with either no edema or minimal edema of both pretibial regions. Consistently, he had manifested a trifle more swelling in his right lower extremity when edema was present.

Within the first month out of the hospital, the patient was vigorously engaging in activity around his home, with no ill-effects. With intentional weight reduction, his weight progressed down to 171 lb. (77.6 kg.) by January, 1956, a loss of 35 lb. (15.9 kg.) in seven months. Clinical examination of the heart, lungs, and blood pressure revealed no abnormalities, and the patient continued to feel better with his weight reduction. His activity increased to where he could be up most of the day with only minimal edema of his lower extremities. He did, however, continue to complain of some "tired feelings" in his feet when he was up for a long time. He stated that he did much better with walking than with standing or sitting around. The cord-like thrombophlebitic veins in the left lower quadrant of the abdomen completely subsided within a period of a few months after his discharge from the hospital and have caused no further difficulty whatsoever, despite his insistence on wearing a belt. As of late 1956, this patient returned to his former occupation.

Comment

This case seemed worthy of reporting because of the unusual nature of the postoperative superficial thrombophlebitic process, which was thought to be a direct result of the ligation of the inferior vena cava. We felt that the constriction of his belt and other garments at the level of the costal margin produced a stasis of blood in compensatorily dilated abdominal veins. This subsided nicely on the usually accepted therapy of bed rest, anticoagulants, and general supportive measures. It was thought at first possibly to be a result of the prostatitis, which he had, but this idea was discarded when a red streak was seen around his entire trunk from the pressure of his pajama cord at the height of the costal margin (the exact level at which the thrombophlebitic process began). Aside from this unusual manifestation as described, we would like to call attention to the patient's clinical course after his loss of weight and utilization of elastic stocking support. He cooperated extremely well with regard to intermittent periods of rest and elevation at first; later these no longer were deemed necessary.

We consider this related to Mondor's disease, although those cases manifest themselves in the breast or chest wall with only some extension down to the costal margin or lower, whereas in this patient it began apparently in the superficial epigastric or superficial iliac circumflex veins or their direct tributaries. It then extended from the costal margin down to Poupart's ligament and did not manifest itself to any extent on the chest wall. However, no mention is made in the cases of Mondor's disease[1] of there being any relation to a previous ligation of the inferior vena cava.

With regard to inferior vena cava ligation, we agree with the general recommendation of the literature that it should be performed only as a life-saving measure as was done in this case. It has been stated[2c] that 85 to 90% of all patients with thromboembolism could be treated successfully with anticoagulants and general supportive measures. With this we agree. The complications from ligation of the inferior vena cava may be severe and significant enough to condemn this procedure as a "routine" one or as mere prophylaxis against thromboembolism.

We are truly unable to state the origin of the thromboembolism and, therefore, do not know that either leg was "involved" although the right thigh did receive the minor laceration and continued to manifest slightly more edema than the left.

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BRONCHIAL ABRASION MICROBIOPSY INSTRUMENT

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The purpose of this communication is to present a modification and improvement of an instrument previously presented,[1] to further its usefulness and adaptability in obtaining material for cytological interpretation from the trachea, main and segmental bronchi.

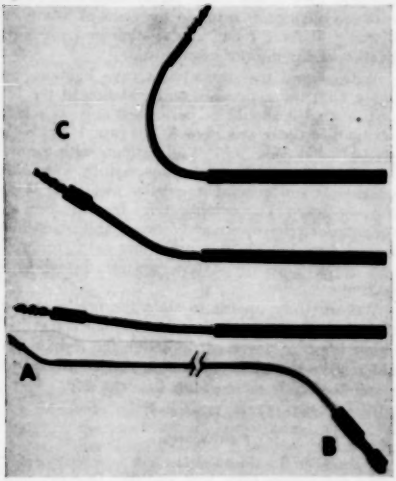

Fig. 1.—Bronchial abrasion microbiopsy instrument: *A*, flexible distal end with abrader (brush); *B*, actuating device; *C*, insert showing flexibility of distal end.

Description of Instrument

The body of the instrument, as seen in figure 1, is made of stainless steel and is 49.0 mm. long and 9.3 mm. in diameter. At the distal end is a screw chuck, attached to the body by a flexible sleeve. A disposable part, a twisted wire-handled nylon abrader (brush), when screwed into the chuck, can be moved from a straight line to an angle of approximately 110 degrees to the long axis of the body. The proximal end of the instrument, bent at an angle of 45 degrees, contains an actuating device which permits the abrader to be rotated about the body of the instrument in either direction, through an angle of approximately 300 degrees. The position of the abrader, with relation to the

Fig. 2.—Microbiopsy specimen positive for cancer from upper lobe bronchus. Roentgenography and bronchial washings were negative. (× 190).

From the Cytology Institute for Cancer Detection and Research.

handle or body, should be determined by rotating the actuating device before the instrument is inserted into the bronchoscope.

Procedure

The bronchoscopic examination is done in the usual way. Abrasion microbiopsy testing is indicated in all cases where there is any possibility of the presence of bronchogenic carcinoma. A separate abrader should be used for each site to be tested, and at least one direct smear made of the abraded material obtained from the trachea, main and segmental bronchi. The specimens, fixed immediately in pure methyl alcohol or in the standard ether-alcohol solution, require no further processing before staining. The use of microscopic slides with frosted ends is recommended. These should be numbered prior to the examination and a paper clip attached or a small rubber band placed about each slide.

Comment

The bronchial abrasion microbiopsy instrument is presented as a new and effective additional aid to early diagnosis of bronchogenic carcinoma. The cellular material obtained with it is actually abraded from beneath the exfoliative level of the mucosa. The cells, being "alive" when detached, stain well (fig. 2). The material is invariably rich in amount and consists of not only single scattered cells but also clusters and groups of cells as well as cohesive particles of mucosa (microbiopsy specimens). This permits better differentiation of malignant changes.

In a case in which the test was repeated after a week's interval, the only cell changes noted were of a mild inflammatory nature.

Bronchogenic carcinoma is the most frequent cancer in men in the United States. The incidence is rising at an alarming degree. Despite the intense clinical studies and the improvement of operative techniques, the five-year survival rate remains deplorably low. It is believed by some authorities[2] that the present rising total incidence of cancer in man seems contributable in large measure to primary cancer of the lung. Cytological methods are proving of great value as diagnostic aids, but there are many obvious reasons why their application is limited.

Abrasion microbiopsy testing can be quickly and easily done. No increased local anesthesia is necessary. In most cases the patients are unaware that the particular test is being done. Occasionally, slight bleeding of a few minutes' duration has been noted.

Summary

A modified and improved abrasion microbiopsy instrument, useful in obtaining material for cytological interpretation from the trachea, main and segmental bronchi, is presented. It has been designed for use through a bronchoscope and is recommended when there is any question of the presence of bronchogenic carcinoma. It eliminates the use of repeat roentgenography when cytology is positive, and reduces the amount of roentgen-ray exposure in suspect cases. With it, correct diagnoses of malignancy have been made in cases where all other methods failed (fig. 2). In some cases, where the roentgenography was either positive or suggestive of malignancy, and the abrasion study was negative, the correctness of the latter diagnosis was sustained either by exploratory operation or by the subsequent clinical course.

135 E. 65th St. (21).

This instrument was part of a Scientific Exhibit at the American Medical Association Convention, New York, June, 1957.

Mr. Jack Harris, consultant on instrumentation, Department of Biology, New York University and the Cytology Institute for Cancer Detection and Research, built this instrument.

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LEG INJURIES IN CHILDREN.—In the examination of injured infants and young children up to the age of four or five years, clinical diagnosis as to site of injury may be quite unreliable. For this reason, a much larger area should be included in the film study, particularly when extremities are involved. It is a frequent experience to have a child of two or three years referred for roentgen study of the hip or upper leg because he refuses to walk after an injury. Many times, a fracture in the lower end of the tibia will be found if the entire leg is included on the films. Even when no fracture is seen, it is obvious that a significant injury has occurred if the young child will not walk or use an extremity. In this situation, it is important that the radiologist define the limitations of the roentgen examination to the referring physician and suggest that re-study be considered if the symptoms continue for a week or ten days. If this is done, some of these patients will show early callus formation at the site of an incomplete greenstick fracture that could not be seen on the original films.—R. P. Barden, M.D., Bread and Butter, editorial, *Radiology*, December, 1957.

DIAGNOSTIC PROBLEMS

HEART FAILURE, FEVER, AND SPLENOMEGALY IN A MORPHINE ADDICT

Clinical Pathologic Conference (PM 566-57), Oct. 10, 1957, from the Department of Pathology and the Hektoen Institute for Medical Research of Cook County Hospital, Chicago, presented for publication by Daniel S. Kushner, M.D., and Paul B. Szanto, M.D.

Clinical Data

A 40-year-old man entered Cook County Hospital on March 23, 1957, complaining of progressive exertional dyspnea, chest pain, mild orthopnea, intermittent ankle edema, night sweats, marked weight loss, and enlargement of the abdomen of three months' duration. For one month, he had had rhinorrhea, sore throat, mild cough, chills, fever, weakness, and epistaxis. He had also complained, since childhood, of urinary frequency (every 45 minutes), nocturia (15 to 20 times), polydipsia, dysuria, and urinary dribbling.

His history revealed gonorrhea at the age of 17 and jaundice at the ages of 17, 23, and 37 years. He was divorced, lived alone, and had worked as a waiter and bartender. He smoked one pack of cigarettes daily, and in the past had used alcohol heavily. He had been a morphine addict intermittently for 17 years, taking 30 mg. three times daily intravenously, but he had not taken morphine for three and a half months prior to admission.

He appeared to be chronically ill, thin, and pale. His blood pressure was 130/85 mm. Hg, pulse 108 per minute, respirations 28 per minute, and temperature 98.6 F (37 C). He was mildly orthopneic. There was mild conjunctival icterus, and an ulcerated area with bleeding points on the left side of the nasal septum. Almond-sized, discrete, tender lymph nodes were noted in the axillas, and pea-sized nodes were present in the cervical and inguinal regions. The lungs were clear. The heart was not enlarged. A systolic murmur was present at the base. The second aortic sound was louder than the second pulmonic sound. The abdomen was protuberant. The liver was tender, extending 5 fingerbreadths below the costal margin; its surface was suggestively nodular. The spleen, also tender, extended 7 fingerbreadths below the costal margin, almost to the iliac crest. Ascites was present. Rectal examination revealed a small prostate, brown stool, and a negative benzidine test. Examination of the extremities revealed 3+ pitting edema, muscular weakness, and the characteristic needle marks of the narcotic addict. There was no clubbing of the fingers or toes.

Urinalysis revealed specific gravity ranging from 1.010 to 1.016; 0 to 2+ proteinuria, a trace of urobilinogen and 3 to 50 red blood cells, 3 to 20 white blood cells, occasional red blood cell cast, and 2 or 3 granular casts per high-power field. A tuberculin skin test was positive with use of a dilution of 1:100. One blood culture was negative, and a second revealed a yeast-like organism. Ascitic fluid culture revealed Escherichia coli. The arm-to-tongue circulation time was 20 seconds with use of magnesium sulfate. The bone marrow showed moderate hypercellularity; normal megakaryocytes; a nucleated red–white blood cell ratio of 1:1; normoblastic erythropoiesis; markedly toxic granulopoiesis; slight increase in lymphocytes, eosinophils, monocytes, histiocytes, and plasma cells (tables 1 and 2).

After admission, the patient had chills and fever, with temperature ranging to 105 F (40.5 C). Two grams of tetracycline and 100 mg. of cortisone were given daily throughout the hospital stay. He initially became afebrile, but later his temperature ranged from 100 to 100.6 F (37.7 to 38.1 C). Additional therapy consisted of vitamins, meralluride (Mercuhydrin) sodium, and supplemental doses of potassium chloride. The patient's edema persisted. The spleen slowly became smaller and less tender, but the hepatomegaly persisted. One week before death, paracentesis revealed clear fluid with a specific gravity of 1.010 and a protein content of 0.5 Gm. per 100 ml. The next day petechiae were observed over the upper extremities. Corticotropin infusions, 40 units daily, were begun, and 2 Gm. of streptomycin and 400 mg. of isoniazid were given daily. The day before death, tonic convulsions involved the face and right side of the body; the pupils were dilated; he bled from the gums and tongue, and had coffee ground emesis. He lapsed into coma, and died on April 28, four months after the onset of illness.

Clinical Discussion

Dr. S. Howard Armstrong Jr.: This unhappy patient was a bartender, who had used morphine intermittently for 17 years. In this hospital heroin

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addicts are more commonly seen than morphine addicts. The heroin addict is usually admitted with hepatitis or tetanus, and most are dead long before their 30th year. Morphine is a comparatively difficult drug to use because its potency is about one-tenth, milligram for milligram, that of heroin. Hospital personnel are particularly susceptible because of the ready availability of the drug. My own experience with morphine addiction comes from teaching hospitals where, in the last 20 years, I have had partial responsibility for four addicts, all of whom were members of the house staff. Of these, two are completely rehabilitated, one is not rehabilitated, and one has been lost sight of. One patient was withdrawn from morphine by substitution of codeine.

No one has yet succeeded, despite intensive efforts in synthetic organic chemistry, in dissociating the addictive from the analgesic and other effects of morphine.[1] Many synthetic substances still retain addictive properties, even demonstrable in tissue culture, indicating their profound biological effects, but some of the drugs of the methadone group have a different ratio of addictive to analgesic effect. The clinical entity of morphine addiction and the withdrawal syndrome and its therapy have been thoroughly reviewed.[2] Turning back to the patient under consideration, three months before admission he had symptoms characteristic of cardiac failure: dyspnea, orthopnea, edema, and exertional chest pain. His heart size and blood pressure were normal, and there was no evidence of valvular disease, except for the description of a basal systolic murmur. The second pulmonic sound was not accentuated, which is of some interest in the light of experimental chronic morphine intoxication in monkeys leading to organic pulmonary changes, but this has not been observed in human beings. The circulation time was prolonged.

Dr. Jacob W. Fischer: The electrocardiogram shows low voltage but T waves of good amplitude. I would conclude that the electrocardiogram is normal.

Dr. Armstrong: There is a marked discrepancy between the three-month-old symptoms of cardiac failure and the relatively negative cardiac examination. In addition, another group of symptoms of one month's duration consisted of running nose, nosebleeds, sore throat, cough, chills, fever, and malaise. Rhinorrhea, excessive lacrimation, shakiness, weakness, malaise, and chilly sensations are classic narcotic withdrawal symptoms, but sore throat and cough demand another explanation. On physical examination, the emaciation could be attributed to long-standing narcotic addiction, especially if combined with chronic alcoholism. No pupillary abnormalities were described, suggesting that he had not had morphine just before admission, since there is no development of pupillary tolerance to chronic morphine administration. Nothing was found in the pharynx or lungs to account for the sore throat and cough.

No mention was made of nodular masses palpated in the left colon as one might expect to find in the chronically constipated morphine addict. However, both liver and spleen were very large and tender, and ascites was present. Too little emphasis is given generally to the relationship between cardiac and hepatic disease and narcotic addiction. Morphine addiction, and in fact any intravenous drug addiction, is associated with four infectious diseases: malaria, tetanus, bacterial endocarditis, and viral hepatitis. Malaria may be needle-transmitted from other addicts who harbor active plasmodia; this situation is becoming rare in the United States. Tetanus is more frequent with heroin addicts who are careless about needle sterilization. Subacute bacterial endocarditis is a well-known complication of morphine addiction. The problem arises whether endocardiosis or rheumatic valvulitis is always underlying or whether the endocarditis can be superimposed on entirely

TABLE 1.—*Hemogram*[*]

Date	Hemoglobin, %	Erythrocytes, Million per Cu. Mm.	Leukocytes, per Cu. Mm.	Platelets	Neutrophils, %	Band Cells, %	Eosinophils, %	Basophils, %	Lymphocytes, %	Monocytes, %
3/25	56	3.28	5,300	decreased	61	18	1	1	19	0
3/28	69	3.98	4,250	decreased	66	25	0	0	2	7
4/ 9	51	2.99	3,100	OK	57	9	0	0	17	17

[*] Smears revealed: 1+ anisocytosis, poikilocytosis, polychromatophilia, toxicity, and occasional burr cells. No malarial parasites observed. Hematocrit 32%, prothrombin time 21 seconds (29% of control), platelet count: 10,000 per cubic millimeter (3/27); 170,100 (3/29); 310,440 (4/2); and 90,000 (4/30).

TABLE 2.—*Summary of Biochemical Data*

Laboratory Values	3/25	3/28	4/5
Nonprotein nitrogen, mg./100 ml.	27		32
Total protein, Gm./100 ml.	6.8	7.0	6.0
Albumin, Gm./100 ml.		2.4	2.5
Globulin, Gm./100 ml.		4.6	3.5
Cholesterol, mg./100 ml.	67		
Cholesterol esters, %	24		
Alkaline phosphatase, Bodansky units			4.0
Icterus index, units	17	13	
Cephalin flocculation	4+		4+
Thymol turbidity, units	20.0		13.8
Gamma globulin, Gm./100 ml.	4.67	3.30	2.39
Diastase, units	8		
Mucoprotein		79	
Chloride, mEq./Liter			95.0
Sodium, mEq./Liter			128
Potassium, mEq./Liter			3.3

normal heart valves. One reported case of endocarditis in a morphine addict occurred on an arteriosclerotic valve.

The most common complication of intravenous injection of drugs is viral hepatitis. This patient had both alcoholism and three previous episodes of jaundice. His liver was enlarged, tender, and nodular, raising the strong presumption of postnecrotic cirrhosis. An elevated level of serum gamma globulin, positive cephalin flocculation, high thymol turbidity, and normal or low mucoprotein level are characteristic of postnecrotic cirrhosis.

Dr. William Meszaros: The posteroanterior roentgenographic view of the chest showed normal heart size and configuration. The right lung was clear. Neither hilar shadow was prominent. A hazy density in the left middle and left lower lung fields suggested pneumonitis. The abdominal film showed a large mass in the left upper quadrant with the configuration of spleen. The liver shadow was not well outlined, but a hazy density throughout the abdomen was compatible with ascites. The intravenous pyelogram showed the outline of the left kidney and collecting system; it was displaced downward but was not part of the mass, confirming it as spleen.

Dr. Armstrong: The ascitic fluid contained only 0.5 Gm. per 100 cc. of protein and was, therefore, a transudate, suggesting portal hypertension as the primary cause for the ascites rather than an inflammatory process. Subacute or acute bacterial endocarditis could explain the development of chills, fever, petechiae, and cardiac failure. Coronary seeding by bacteria from infected heart valves can be associated with diffuse septic myocarditis, or aneurysm of the aortic sinus; either could easily explain the congestive failure and even exertional angina. Thus can one explain the development of cardiac failure in the absence of functionally significant valvular disease. From one of two blood cultures, a yeast-like organism was isolated.

Dr. William Metzger: The organism could not be identified as a pathogenic yeast. On the basis of the frequency of yeast-like skin contaminants in blood cultures, I would regard it as a saprophyte.

Dr. Paul B. Szanto: What about Candida albicans?

Dr. Metzger: It could not be identified as a Candida species.

Dr. Armstrong: Persons in occupations which require keeping their hands in water get Candida infections; this includes housewives and also bartenders. Monilial endocarditis has been observed in morphine addiction. This bartender could have gotten a paronychia infected with Candida with which he might have contaminated his venous blood in the process of self-injection with morphine. The therapy with tetracycline diminished but did not eradicate his fever, although his chills abated. Although he was given cortisone, which could have facilitated spread of infection in the absence of available specific antifungal or antibacterial therapy, he did improve somewhat. Eventually a systemic dissemination of infection did occur. One could relate his leukopenia to the hypersplenism of cirrhosis. Because the spleen was tender, I would expect splenic infarcts, embolic from the infected heart valves, and not just the congestive splenomegaly of cirrhosis. The intermittent microscopic hematuria I would relate to embolic focal glomerulonephritis. Terminally the focal convulsions suggest cortical brain hemorrhage. My diagnosis is chronic hepatitis, with postnecrotic cirrhosis, and subacute bacterial (or fungal) endocarditis.

Dr. Frederick Steigmann: This patient had only one chill and the high temperature lasted only two days. Not infrequently, especially in elderly patients, so-called cardiac symptoms in cirrhotics are in reality related to severe anemia. Several things point to the diagnosis of postnecrotic cirrhosis, especially the episode of jaundice at the age of 17, before he was a narcotic addict or an alcoholic. Also in postnecrotic cirrhosis the spleen is frequently much larger than the liver, as opposed to nutritional cirrhosis. I wonder about the large glands in the axilla.

Dr. Armstrong: They were only almond-sized and may have been nonspecific.

Dr. Benjamin Gasul: It is time that we discarded the comparison, "The second aortic sound was louder than the second pulmonic," because there is no such thing as a second pulmonic sound. In the aortic area, a single sound is heard due to closure of the aortic valve. In the pulmonic area, two sounds are audible due to closure of the aortic valve followed by closure of the pulmonic valve. Therefore, the correct statement should be, "The second component of the pulmonic second sound is accentuated."

Dr. Louis Feldman: There is a possibility that the right side of the heart was involved by the endocarditis, producing bacterial emboli in the lung. Since the bacteria would be filtered out by the pulmonary capillaries, blood cultures would be negative.

Clinical Diagnosis.—The clinical diagnosis was subacute bacterial (or fungal) endocarditis and postnecrotic cirrhosis.

Pathologist's Report

Dr. Szanto: The three roads which lead the heroin and morphine addict to the pathologist (tetanus, viral hepatitis, and bacterial endocarditis) have already been discussed. Inspection of the body revealed no evidence of malnutrition. Numerous petechiae and ecchymoses were scattered over the skin. A few petechiae were also seen in the conjunctivae. A large mass was palpable in the left upper abdominal quadrant. There was pitting edema of the lower extremities. The peritoneal

cavity contained 2,500 cc. of serous fluid. Numerous ecchymoses of the serosal surfaces were present. The left pleural cavity contained 450 cc. and the right 200 cc. of serous fluid.

The repeated episodes of jaundice from the age of 17 may have resulted from a viral hepatitis leading to postnecrotic cirrhosis. The liver was enlarged (1,900 Gm.), firm, and diffusely nodular. The nodules were generally uniform in size, although larger nodules were present on the inferior surface. The uniform fine granularity speaks for Laennec's (septal) cirrhosis, but the large nodules on the inferior surface would support the clinical diagnosis of postnecrotic cirrhosis. On the basis of gross appearance, this would appear to be a postnecrotic cirrhosis with transition into Laennec's cirrhosis. Cut sections revealed uniform nodularity and connective tissue septums dissecting the normal parenchyma. Histologically, extensive reconstruction was present. The absence of normal portal fields within the large nodules and the absence of completely collapsed lobules in broad bands of connective tissue may be used as arguments against the diagnosis of postnecrotic cirrhosis. It is possible that the patient originally had postnecrotic cirrhosis developing from a viral hepatitis, with subsequent changes aggravated by nutritional deficiency, secondary to alcoholism, presenting terminally the gross and microscopic appearance of Laennec's (septal) cirrhosis. This cirrhosis may at least partially account for the severe ascites. Large esophageal varices in the lower part of the esophagus and cardia of the stomach confirm the presence of portal hypertension of a far-advanced hepatic cirrhosis.

Portal and splenic veins were free of thrombus. Moderate pancreatic fibrosis was present, as is seen in cirrhosis, as a result of nutritional deficiency. The spleen was markedly enlarged (1,750 Gm.), extending 10 cm. below the costal arch. A few anemic infarcts explain the left flank tenderness. The microscopic appearance of the spleen differed from that seen in chronic congestive splenomegaly. The typical fibrocongestive splenomegaly of hepatic cirrhosis is characterized by a pseudoglandular appearance, with wide sinuses lined by swollen endothelial cells and fibrosis of the intersinusoidal cords. In this case, the sinusoids were engorged and there was no evidence of fibrosis. The follicles were small, and they showed prominent central arteries. Numerous plasma cells and polymorphonuclear leukocytes were present in the red pulp. This was a subacute infectious splenitis, which may have been superimposed on a preexisting fibrocongestive splenomegaly. The degree of splenomegaly in cirrhosis is variable but not as great as is often assumed. In a survey in our department of 1,426 cases of cirrhosis, the spleen weighed less than 300 Gm. in 59% of the cases; between 300 and 600 Gm. in 32.5%, and over 600 Gm. in 8.5% of the cases. Therefore, to explain such marked splenomegaly as was present in this case, we must consider some factor such as infection or lymphoma.

The clinically enlarged superficial lymph nodes contributed to the diagnostic difficulties. Microscopically, they showed only chronic lymphadenitis, as is frequently found in narcotic addicts. The bone marrow in the diaphysis of the long bones was red, and microscopically showed marked reactive erythromyelopoietic activity. The lungs contained foci of septal and interstitial fibrosis, the appearance of chronic pneumonitis. A healed apical tuberculosis was also present.

Another possible focus of infection explaining the chronic splenitis could be the heart. The heart was not enlarged (350 Gm.). Prominent trabeculae of the right ventricle, and the presence of pulmonary

Fig. 1.—Large vegetation on atrial surface of mitral valve between two leaflets.

sclerosis pointed toward increased pulmonary arterial pressure, perhaps related to the chronic pneumonitis. The left atrium was normal, as was the circumference of the mitral valve ring (12 cm.). The mitral leaflets and chordae tendineae were diffusely thickened. Superimposed on this old healed process, on the atrial surface of the valve, were two equal-sized, moderately friable, brownish, granular vegetations (fig. 1). Histologically, the superficial layer of the vegetation consisted of fibrin with some leukocytes. Acute and chronic inflammatory cells were present beneath this layer, as seen in so-called subacute bacterial endocarditis. In some areas, gram-positive organisms could be demonstrated on the surface of the vegetation. Whenever we find bacteria on the surface of a vegetation, we would expect that blood cultures

taken during life should be positive. This emphasizes the importance of intensive expert bacteriological studies of repeated blood cultures in patients suspected of having bacterial endocarditis. Anaerobic as well as aerobic cultures should be made and observed for several weeks before discarding. The myocardium showed old spindle-shaped scars

Fig. 2.—High-power view of vegetation, showing clusters of yeast cells.

and evidence of a more recent moderate interstitial myocarditis, findings which typically accompany endocarditis.

The classic sharp distinction between acute and subacute bacterial endocarditis is not justified, because of the changing anatomic picture under the influence of antimicrobial therapy. An etiological classification is preferable, since modern antimicrobial therapy may modify the natural history of endocarditis.

In addition to the gram-positive organisms, one section through the vegetation on the mitral leaflet showed clusters of fungi (fig. 2), which could be classified with the Candida species, although no cultural confirmation was possible. A heroin or morphine addict may develop mycotic endocarditis, usually due to Candida albicans. In my opinion, this patient originally had bacterial endocarditis and developed a superimposed mycotic endocarditis.[3]

The target organs involved in bacterial endocarditis are the kidney, spleen, brain, and skin. The kidneys weighed 350 Gm. together, and showed a few distinct petechiae on their surfaces. Interstitial infiltration by inflammatory cells and segmental fibrinoid degeneration of a few glomeruli suggested focal "embolic" glomerulonephritis (fig. 3). A few anemic infarcts were also seen. Focal glomerular lesions are usually associated with microscopic hematuria, while renal infarction more often leads to gross hematuria. It must be emphasized that the microscopic hematuria of endocarditic focal glomerulonephritis is typically transient. Therefore, one must daily examine the urinary sediment, because the finding of transient microscopic hematuria is almost as pathognomonic as a positive blood culture. The focal "embolic" glomerulonephritis is not due to bacterial emboli, but is a fibrinoid necrosis, presumably related to a hyperimmune state developing due to continued or transient bacteremia. As a result of the fibrinoid necrosis, the capillaries rupture, the erythrocytes reach Bowman's space, and eventually hematuria occurs (fig. 4).

A patient with subacute bacterial endocarditis may have either infarcts or focal embolic glomerulonephritis. In this instance, both were present.

Fig. 3.—Focal fibrinoid degeneration of glomerular tufts ("focal embolic glomerulonephritis").

Diffuse glomerulonephritis, which infrequently occurs in bacterial endocarditis, was not present in this case.

This patient had tonic convulsions, and correspondingly perivascular cuffing, foci of microglia cells, and microabscesses were found in the subcortical white matter of the brain. Some vasculitis with focal hemorrhages was seen in the same areas. In addition, a massive cerebral hemorrhage practically destroyed the left frontal and occipital lobes (fig. 5), resulting in a subarachnoid hemorrhage.

No mycotic aneurysm, as characteristically may occur in bacterial endocarditis, and the rupture of which could account for the massive hemorrhage, was present in this case. The described microscopic vasculitis may account for this cerebral hemorrhage, but its massive character suggests that the thrombocytopenia related to hypersplenism may have played a role in the precipitation or aggravation of the hemorrhage.

Fig. 4.—Hemorrhage into Bowman's space (leading to microscopic hematuria) secondary to fibrinoid degeneration.

Pathological Diagnosis.—The pathological diagnosis was bacterial and mycotic endocarditis superimposed on fibroplastic deformity of the mitral valve; focal endocarditic glomerulonephritis; multiple renal infarcts; Laennec's cirrhosis; splenomegaly (due to fibrocongestion and chronic splenitis); and massive cerebral hemorrhage.

Comment

Dr. Szanto: This patient had viral hepatitis many years ago. He was a chronic alcoholic and developed cirrhosis, which may have been originally of postnecrotic type with transition into Laennec's (septal) cirrhosis. He developed bacterial endocarditis, one month prior to his death, possibly secondary to pneumonia. Superimposed on the bacterial endocarditis, he developed a systemic mycotic infection, resulting in mycotic endocarditis. The red blood cells in the urinary sediment were due to focal embolic glomerulonephritis and,

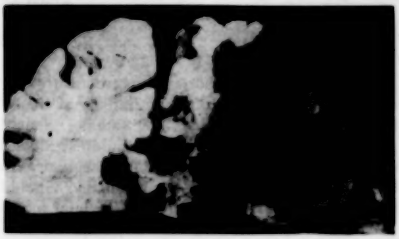

Fig. 5.—Massive hemorrhage destroying right frontal lobe, leading to subarachnoid hemorrhage (horizontal section).

finally, the terminal convulsions were due to massive hemorrhage secondary to thrombocytopenia, and not due to bacterial emboli.

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A NEW VIRUS.—The agent described in this paper is considered to be a newly-recognized arthropod-borne virus. Infection with this virus seems to be fairly prevalent in C. tarsalis in California, the recovery rate of the virus being approximately 3 per cent of all the pools tested. Infection is not restricted to C. tarsalis, however, since three strains were recovered in 1956 from Culex stigmatosoma. With respect to C. tarsalis, evidence of infection in this species appears during June or early July, the infection rate becomes maximal during July and thereafter diminishes, to fall to nondemonstrable levels by October. . . . The role of the Turlock virus, if any, in the causation of human or animal disease has yet to be determined. Information thus far acquired, and based on limited material, suggests that human infection with the agent is not common. Likewise, no evidence was obtained that this virus is etiologically involved in the human encephalitides.—E. H. Lennette, M. I. Ota, F. Y. Fujimoto, A. Wiener, and E. C. Loomis, Turlock Virus: A Presumably New Arthropod-Borne Virus: Isolation and Identification, *The American Journal of Tropical Medicine and Hygiene*, November, 1957.

COUNCIL ON FOODS AND NUTRITION

Report to the Council

The Council has authorized publication of the following report. This article is part of a nutrition education program which is assisted by the Nutrition Foundation, Inc.

PHILIP L. WHITE, SC.D., *Secretary*

FEEDING OF INFANTS AND CHILDREN IN HOT WEATHER

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When the weather is hot, parents frequently consult their physicians as to the desirability of changing the dietary patterns of their families. Should salt be added to the diets of their children? Should certain foods be prohibited? Is it necessary to change the food mixture for the baby? Should modifications be made in the diets of infants and children when the environmental temperature is high? Do special psychological problems related to eating arise during hot weather? There is little scientific data concerning the effects of climate on the dietary requirements of infants and children, but pediatricians practicing in areas where prolonged periods of hot weather occur have amassed a considerable body of experience. This report combines these sources of information.

Analogies drawn from the food habits of the native populations in tropical or desert regions are not helpful, because the native diets are largely determined by factors of availability, social customs, and economics, rather than by those related to health or comfort. In general, prosperous individuals in such areas gradually assume diets similar to those of well-to-do people living in temperate zones.

Nutrient Requirements

Proteins, Minerals, and Vitamins.—The requirement for protein seems to be unchanged or possibly is increased in hot weather.[1] When sweating is profuse, there are probably increased skin losses of water-soluble vitamins, calcium, and iron. A decade ago the beneficial effects of increased vitamin C or thiamine intake on work performance in hot weather were promoted in the popular press. Subsequent studies, more carefully controlled, have failed to demonstrate any benefit in biochemical, psychomotor, or physiological functions or in protection from heat exhaustion.[2] Thiamine and vitamin C requirements or rates of depletion of these vitamins reveal no differences between summer and winter values.[3] More adequate data concerning children are needed. However, clinical experience would seem to support the conclusion that the preponderance of evidence from human experiments indicates that the vitamin requirements are not increased by residence or by work in a tropical climate.[1] Hot weather appears to impose no special dietary requirements [4] other than the needs for salt and water.

Sodium, Chloride, and Water.—As the environmental temperature approaches skin temperature, the evaporation of water plays an increasing role in heat dissipation. All the loss of heat from the body is accounted for by the evaporation of water from the lungs and skin at temperatures above that of the skin. The amount of water so lost is related to total heat production, to any gain of heat from the environment by radiation, conduction, or convection, and to the temperature and humidity of the surroundings. Sweat losses averaging 56 cc. per kilogram of body weight per 24 hours were measured from nude infants in a room kept at a temperature of 91 F and at low relative humidity.[5] Sweat losses as high as 100 cc. per kilogram of body weight per day were found in infants ill with diarrheal disease who were studied in nonairconditioned wards in Galveston and Dallas, Texas.[6] Under severe conditions adults may lose 5-10 liters of water as sweat per day.

Water deficits as low as 1-2% of the body weight may produce measurable adverse effects on the

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circulation of blood and the body's tolerance to heat.[7] The greatest efficiency is achieved by both infants [8] and adults [1] when water losses are replaced hour by hour. However, men and children who are acclimated seem to accumulate slight water deficits voluntarily during the day, but they make up these deficits at mealtimes and during the evening.[7]

The concentrations of sodium and chloride in sweat are dependent on numerous factors.[8] In healthy individuals acclimated to heat, the concentrations of sodium and chloride are quite low. In healthy infants, these concentrations, calculated from total skin washings and the estimated volumes of sweat, averaged 4.8 mEq. of sodium and 6.1 mEq. of chloride per liter of sweat.[3] Although the concentrations of these electrolytes in sweat may be as low in adults, they are often three to five times as high.[7] Studies of both infants and adults indicate excessive sodium chloride intake to be deleterious to the adjustments to heat stress; thus, it should be avoided.[8] The intake of sodium and chloride in the ordinary diets of healthy infants appears to be adequate, judging from extensive practical experience and from the scanty data available. If table salt is used in ordinary amounts in the preparation of foods for acclimated older children and adults and is available to them to be added at the table according to desire, there appears to be no need for specifically prescribing additional sodium chloride, either as tablets or as treated drinking water, except under conditions of great physical activity associated with extremely large outputs of sweat.

It should be clear that these statements concerning the adequacy of dietary sodium and chloride apply to healthy infants and children maintaining normal intakes. Cardullo,[10] Evans,[11] and Kessler and Anderson [12] have discussed the adverse effects of heat on hospitalized infants and children. The administration of sodium and chloride was clearly of benefit to those patients who had "salt" depletion due to diminished intake in hot weather because of infection, recurrent vomiting, or injuries. Depletion was also caused by losses of electrolytes from the gastrointestinal tract or by abnormally high concentrations of sodium and chloride in the sweat of patients with pancreatic fibrosis. The frequency and seriousness of hyperpyrexia during hot weather in infants with cerebral damage is stressed by Cardullo.[10] The administration of added sodium chloride to the formulas of healthy infants did not alter their course, but some refused the "salt feedings." [10]

Although it is implied that the addition of sodium chloride to the diet might be useful for healthy infants, no evidence has been presented to support this contention. Practical experience indicates that whenever an infant or child on a usual diet suffers from heat stress, the likelihood is great that the individual is ill and merits further investigation, particularly for pancreatic fibrosis, adrenal insufficiency, or incipient diarrheal disease.

Eating Habits and Psychological Problems

The practitioner accustomed to the problems of children's eating habits during hot weather finds no great difficulties. The observed changes in eating and living habits are related to attempts to keep cool. Children appear to reduce their caloric intake and diminish their rate of weight gain, while maintaining their rate of gain in height in warm weather. There is a strong desire to drink and eat cold liquids and foods, some of which, like ice cream, may be high in calories. When children are at active outdoor play, they need a considerable fluid intake. If parents permit, many children will drink two or more quarts of cold milk or sweetened carbonated beverages. Even cold water, if taken shortly before meals, appears to suppress the appetite temporarily, thus producing poor mealtime habits or attitudes.

During recent summers a "new syndrome" has appeared. It is characterized by the pale, flabby, tired child who has gained excessive weight during the warm weather because he has stayed indoors in an airconditioned house watching television most of his waking hours and has indulged in frequent between-meal snacks that have spoiled his appetite for well-balanced meals.

Practical Management

An adequate fluid intake should be assured for infants by regularly offering them a few ounces of water between feedings. In artificially fed infants, it may also be desirable to reduce the caloric concentration of the food mixture to approximately 18 calories per ounce during hot weather.

Individualized management of children during the summer is the best assurance of maintaining good nutrition. However, the following guides are those most often required.

1. The habit of vigorous outdoor activity should be continued or developed. Children do not mind hot weather unless they are conditioned by their elders to dislike it.

2. Cool, but not cold, drinks are best, and, for the most part, water should be used to quench thirst. Evening intake of water should not be unduly limited.

3. Between-meal foods and high-caloric drinks may have to be controlled.

4. A short "cooling-off" and quiet period before meals may increase the child's appetite.

5. Limiting of high-caloric foods, such as peanut butter and ice cream, may be necessary. For some children, the use of skimmed milk may be advisable to promote a better appeite for other foods.

6. The large and better balanced meal may best be served in the evening when the temperature is lower.

7. All of the usual measures and guides for developing good eating habits should be continued, irrespective of the weather.[13]

Summary

Hot weather imposes no special dietary requirements for healthy infants and children, except for an increased water intake. If infants and children ingesting well-balanced diets do not tolerate ordinary heat stress, they should be investigated for illness rather than changing their diets. Poor appetites and faulty eating habits may result from the uncontrolled use of cold, high-caloric drinks or foods, from failure to take adequate outdoor exercise, or from overindulgence in between-meal snacks. It is unwise for adults to "condition" children to dislike hot weather or to foist summertime food fads on them.

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HAZARDS OF OVERTRANSFUSION.—Excess transfusions do not produce arterial hypertension; rather, they produce overfilling and, after failure has begun, hypotension. The differentiation of hypotension due to inadequate transfusion from the hypotension due to left heart failure will be the duty of the physician officiating at the transfusion. The clinical picture of overloading of the left heart is that of acute left heart failure with pulmonary edema. If the patient is awake, he will become restless, have respiratory difficulty and dyspnea. The jugular veins and other peripheral veins are distended, venous pressure is elevated, and the patient will have a plethoric appearance. As pulmonary edema develops, this color tends to become cyanotic in tinge, the respiratory rate increases, audible rales are noted and frothy tracheal mucus may be coughed up, or will appear in the endotracheal tube. Increase in pulse and progressive hypotension will be encountered. The treatment depends in some degree upon the extent of the overtransfusion. Rapid removal of the excess blood must be carried out to remove the strain on the heart. To this end, usually 500 cc. of blood is withdrawn into a transfusion bottle. It is probably not wise to withdraw more than this at one time. Rather, one should observe the response to this one bloodletting, which may be dramatic. The heart may adjust and recover within minutes. Rarely a second bloodletting will be needed. —V. L. Guynn, M.D., and J. T. Reynolds, M.D., The Use and Abuse of Blood Transfusions, *The Surgical Clinics of North America*, February, 1958.

COUNCIL ON INDUSTRIAL HEALTH

The following report has been prepared by the Committee on Occupational Cancer of the Council on Industrial Health.

B. Dixon Holland, M.D., Secretary

OCCUPATIONAL CANCER

METHODS OF EPIDEMIOLOGICAL STUDY

The first form of cancer with an identified causal factor was described nearly two centuries ago. It occurred with unusual frequency among workers in a single occupation and, hence, became known as chimney-sweep's cancer. In spite of the fact that the first knowledge of the etiology of cancer can be traced to Percival Pott's [1] observation on chimney-sweeps, we have no reliable information today from which a reasonable estimate can be made of the proportion of cancers that have some causal relationship to an exposure of occupational or industrial origin. Although a number of specific types of cancer have been associated with occupational exposures since Percival Pott's original studies, there is no reason to believe that these exhaust the possible list of such cancers.

In recent years, an increasing amount of scientific literature has appeared on the subject, confusing, at times, both the physician in private practice and the full-time industrial physician. Part of this confusion arises from the fact that occupational cancer is different from many other well-known occupational diseases, more difficult to identify, and, hence, more difficult to study. Experience has demonstrated that the identification of occupational or industrial causes of human cancer is made difficult by certain characteristics of cancer itself, as well as by labor mobility. Occupational cancers can rarely be distinguished on morphologic grounds from those due to other causes. Furthermore, several years may elapse between the date a worker is first exposed to an agent capable of causing cancer and the time of development of a tumor to the stage where it can be clinically recognized.

From a study of 300 cases of mule-spinner's cancer reported in England and Wales from 1920 to 1928, Henry [2] found that the time elapsing from the date of starting work as a mule spinner until the date of diagnosis of cancer ranged from 16 to 63 years and that for 67% of the cases this latent period varied between 35 and 54 years. Several of the workers were no longer employed in this occupation at the time of diagnosis. The average latent period for mule-spinner's cancer is appreciably longer than that for some other forms of occupational cancer. However, a latent period of several years is characteristic of most cancers. For example, the average interval from initial exposure to diagnosis of lung cancer among workers in United States chromate-producing plants was 17.2 years, with a range of 4 to 47 years.[3]

Finally, there is a "normal" or "expected" incidence of cancer, whereas most occupational diseases do not "normally" exist. Thus, if a man develops silicosis, it probably arose as a result of his occupation, since this disease does not have a "normal" incidence. Accordingly, it becomes necessary in the study of occupational cancer to establish that the disease occurs either at a higher than normal incidence in a given occupation or with a greater relative frequency of site or type, so that there is no doubt about the causal relationship between the disease and the occupation. For this reason, single case reports of silicosis, lead poisoning, or other occupational diseases may make significant contributions to our understanding of most occupational diseases, but individual case reports really contribute little to our understanding of occupational cancer. The same problems have arisen in attempts to relate trauma to cancer—and for the same reasons. These difficulties have led to vigorous debates in the scientific literature, which have perhaps been best summarized by Stewart.[4]

The past three decades have witnessed the birth and large-scale development of experimental methods for the study of carcinogenesis. This has permitted identification of an extensive list of chemical compounds capable of causing cancer in animals in specific experimental situations.[5] A number of these compounds are encountered in various manufacturing processes. It is not correct to assume from these two facts that the risk of cancer developing

Chairman of the Committee on Occupational Cancer is G. Burroughs Mider, M.D., associate director in charge of research, National Cancer Institute, Bethesda, Md. Members of the Committee are as follows: Harold F. Dorn, Ph.D., Biometrics Branch, Division of Research Services, U. S. Public Health Service, Department of Health, Education, and Welfare, National Institutes of Health, Bethesda, Md.; John G. Downing, M.D., Boston; Robert E. Eckardt, M.D., director, Medical Research Division, Esso Research and Engineering Company, Linden, N. J.; Mr. Edward A. Lew, Metropolitan Life Insurance Company, New York; W. Harvey Maguigan, Ph.D., Allied Chemical and Dye Corporation, National Aniline Division, Hopewell, Va.; and John J. Phair, M.D., University of Cincinnati College of Medicine, Cincinnati.

among industrial workers thereby is either increased or left unchanged. This can be ascertained only by appropriately conducted investigations, and the answer ultimately must be derived from observations on workers in specific occupations and industries.

It is essential, therefore, for the investigator who would study occupational cancer, to have, for comparative purposes, reasonably reliable estimates on the normal incidence of cancer of various types and sites. The collection of such data is a difficult and time-consuming task, requiring almost constant work by a team of trained epidemiologists and statisticians. Cancer incidence increases with age, shows site-specific variations with sex and color, may be modified by geographic factors, and is being continually altered by the aging of the population. In addition, certain specific cancers, such as cancer of the stomach in males, may show unpredictable, unexpected, and unexplained changes in incidence with time. Finally, because of improved diagnostic techniques, greater awareness of cancer, changes in the reporting of disease occasioned by legislative or administrative action, and improved reporting of the cause of death, additional artifacts may be introduced into the calculations. At the present time, the best available rates of normal cancer incidence, prevalence, and relative frequency are those assembled by Dorn and associates.[5] Annual and special reports of the National Office of Vital Statistics provide mortality statistics for the entire country and for various geographic subdivisions.[7] Finally, certain states, particularly Connecticut and New York, have assembled similar data.[8] It is to be hoped that such efforts will be continued and expanded in order that up-to-date, reliable figures will be available for the necessary comparisons, not only to aid occupational cancer studies but also to keep abreast of decade-by-decade changes in cancer incidence and distribution.

The physician of today, and increasingly in the future, will be called on to provide answers as to whether the increased use of a chemical or the introduction of a new chemical has increased old hazards or introduced new ones. If the advancement of the chemical age and the anticipated improvements in standards of living continue, it will be necessary for the physician to keep well in mind the caution so well phrased 10 years ago by Stewart[4]: "It is probable that with the development of chemical industry we will see new chemical cancers and medicine must be on the watch for such developments. But it must refrain from hastily ascribing to industry those tumors whose incidence falls well within the expectations for the population as a whole and from making premature conclusions based on lack of appreciation of statistical methods."

While the study of occupational causes of cancer is complicated, it is not impossible. Since the area of investigation is so broad, a great deal of time and energy can easily be spent in fruitless study unless some preliminary idea can be obtained of those occupations which appear to be associated with cancer hazards. The principal support for such ideas or hypotheses is clinical impressions derived from the study of individual cases of disease and the analysis of existing records of morbidity and mortality. Accordingly, it is believed worthwhile to describe various sources of data now available and to indicate, in summary fashion, certain approaches and techniques that may prove fruitful.

Official Statistics

The official certificate of death required for each deceased person in the United States provides for the entry of his usual (and probably last) occupation and industry. Very little use has been made of this source of information, largely due to uncertainty concerning the comparability of the entries for occupation and industry on death certificates with corresponding data from the census of population. Information on this point soon will be available from current study of the corresponding entries for occupation and industry found on the death certificate and on the 1950 census schedule for a sample of deaths.

At best, official mortality statistics provide a measure of the total effect of an occupation and the general level of living of persons following that occupation. Specific occupational hazards are not likely to be identified by a study of these records. Only one entry for occupation is requested, and frequently either no occupation or an entry of "retired" is reported for persons who have stopped working because of old age. Occupations are described only in general terms, so that it is not possible to identify specific groups of workers who are thought to have been exposed to a suspected health hazard. Furthermore, each industry, and usually each individual company, employs entirely different names for the same job assignment.

In spite of their many defects, mortality statistics, if analyzed with imagination, may provide clues to occupational or industrial hazards which can be explored by more specific studies. The following examples illustrate the types of studies that have been made with death certificates.

Mortality Rates by Occupation.—The information on cause of death and occupation and industry on death certificates and census reports can be used to compute death rates from different forms of cancer. The most extensive series of death rates are those published by the Registrar General of England and Wales. References to these data may be found in an article by Kennaway.[9] A comprehensive study of occupational mortality in the United States has never been made. The National

Office of Vital Statistics has initiated a study based upon deaths occurring during 1950, the preliminary results of which have been reported.[10]

Mortality rates by occupation can be computed for the population of specific localities, for example, a city or county, provided the number of workers is large enough to yield reliable rates. The information needed for such studies is (*a*) the number of deaths of persons in each of the occupations to be studied, and (*b*) the total number of persons in each occupation classification by age and sex. The number of deaths can be obtained from the local or state office of vital statistics; unless special information is available locally, statistics concerning the number of workers by occupation must be obtained from the U. S. Bureau of the Census. Studies should be done for a period near the date of the census of population in order to avoid the necessity of preparing population estimates. A good illustration of this approach has been reported from Sheffield, England, by Turner and Grace.[11]

Mortality Rates for Specific Areas.—The analysis of mortality statistics for cancer of specific sites for local areas, such as a county or group of counties, may be undertaken in order to determine whether an unusually high death rate or an unusually large proportion of deaths exists for a specific form of cancer. Available information concerning possible environmental hazards may be studied simultaneously to determine if there are any that appear to be associated with the known facts about cancer mortality.

These studies may be conducted by systematically studying the variation in cancer mortality among the counties of a state, as was done by Mancuso and others[12] in Ohio, or by analyzing mortality from cancer in an area where an industry whose workers are thought to be exposed to some cancer hazard is located. Projects of this kind are most likely to yield useful leads if a large proportion of workers in the area are employed in a single industry. Mortality rates for total cancer and for cancer of specific sites by age and sex can be computed for a period of several years. If the population under observation is too small to yield reliable age and sex specific death rates, an expected number of deaths can be computed by multiplying the population of the local area distributed by age and sex by the death rates for some appropriate standard population, for example, the population of the state in which the local area is located. The expected number of deaths is then compared with the observed number. For comparative purposes, similar computations should be made for other counties that do not have an appreciable number of workers in the industry in question.

If the deaths of persons who have been employed by a specific industry can be identified, a comparison of the causes of death in this group with those of the rest of the population can be made. The study by Hill and others[13] of workers in a factory handling inorganic arsenic compounds is an excellent example of this method.

Mortality from Specific Forms of Cancer.—Since most of the carcinogenic agents of occupational origin that have so far been identified cause cancer of a specific site, several studies have been made in Great Britain, based on death certificates, with a specific type of cancer, for example, cancer of the skin as the cause of death.[14] The usual procedure has been to review all death certificates filed in a specified area for which population data are available and to compute death rates by occupation. The areas selected for study have been either the entire country or a specific local area where the existence of a cancer hazard is suspected.

Social Security Records.—The records maintained by the Bureau of Old-Age and Survivors Insurance, Social Security Administration, Department of Health, Education, and Welfare, include the employment history of all workers covered by the act since January, 1937. Information may be obtained concerning the name and address of each employer, the dates of employment and the industrial classification of each employer according to a four-digit code. These records may be used by themselves or in conjunction with official vital statistics for studies of mortality among persons who have worked in specific occupations.

For example, one could start with a list of persons who are known to have been employed in a given occupation or industry. Although detailed information usually will not be given out for persons who are still living, the date of death and employment history can be obtained for persons who have died. In this way the subsequent history of part or all of the workers who have left the occupation can be traced. Alternatively, one could start with a group of persons who have died from a specific form of cancer and obtain their previous work history. A control group of persons who died from causes other than the type of cancer being studied should be investigated simultaneously in order to judge whether the number dying from cancer who had worked in a given occupation was large enough to suggest the existence of an occupational hazard.

Occupational Disease Reports.—A survey conducted by the United States Public Health Service in 1953 found that only 28 states had statutes or regulations requiring physicians to report patients suffering from occupational diseases. No serious attempt is made to promote compliance, so that the regulations often are ignored.

A much larger number of occupationally induced diseases are reported in connection with claims for workmen's compensation. About one-half of such diseases are skin disorders. Among the remainder, complaints due to physical conditions of the working environment, systemic effects of chemicals, and

infectious diseases are the most common. At best, the claims give only a rough indication of the occurrence of the types of complaints reported. Even where awards have been sustained for occupational cancer, the validity of the alleged causal relationship frequently fails to satisfy critical and competent medical judgment. Neither official reports of occupational diseases nor claims for workmen's compensation have yet yielded useful information concerning the incidence of occupational cancer in the United States.

In contrast, the Factory Inspection System of Great Britain has been an important source of information about occupational cancer. The first medical inspector was appointed in 1898. As presently organized, the medical staff is responsible for the promulgation and supervision of regulations for various occupations, the investigation of cases of industrial poisonings, the study of new industrial processes that may be injurious to health, and the supervision of the work of physicians who give certificates of fitness for work.

A list of lesions that may be caused by known or suspected carcinogenic agents has been established, and these must be reported to the Chief Inspector of Factories. This information is supplemented by inspection of plants where possible carcinogens are produced or handled. Special studies have been made of cancers developing among workers engaged in the extraction, processing, or use of shale oil, petroleum products, asbestos, nickel, and dyestuffs. In 1947, for example, Henry [15] published an analysis of 3,753 skin cancers in 2,975 persons reported to the Chief Inspector of Factories from 1920 to 1945 inclusive. Pitch, tar, or tar-products accounted for about 60% of these cases; shale oil, mineral oil, or bitumen caused most of the remainder.

Insurance Company Statistics

Life Insurance.—Life insurance company follow-up studies have the special merit that they usually deal with relatively large numbers of persons who can be automatically traced for long periods of time through the circumstance of their being insured. A suitable standard of comparison is available in the experience of the aggregate body of insured persons of the particular type. Moreover, in the absence of national mortality statistics by socioeconomic classifications, the experience of different groups of insured persons sheds some light on the relative prevalence of cancer in different socioeconomic classifications.

Life insurance companies maintain detailed records of death claims, disability claims, claims for hospitalization, surgical benefits, and medical expense benefits, as well as special records for medicoactuarial studies and, occasionally, records of periodic health examinations of their employees also. All of these can be tapped for information regarding the prevalence of cancer. It is important to realize, however, that the records maintained by life insurance companies are prepared primarily for administrative purposes and only incidentally yield information about the prevalence of cancer among insured persons.

Generally speaking, from death claim records and records maintained on the insurance in force, it is possible to compute death rates by age, sex, and race for various classes of insured persons. The experience of industrial policy holders of the Metropolitan Life Insurance Company has been reported in a number of studies,[16] which show for the years 1911-1955 the mortality rates by age, sex, and race from cancer of the following sites: digestive organs, female genital organs, breast, buccal cavity, skin, lungs and pleura, bladder, prostate, and other organs. Current information along these lines is published in the Statistical Bulletin of the Metropolitan Life Insurance Company.

Because the applicant's occupation is usually a major factor in determining the life insurance risk, such information is given on the application for insurance. It is often possible, therefore, to calculate the cancer mortality among insured persons by industry or occupation. However, occupation is usually available only to the insurance company at time of application for insurance or at time of death, intermediate occupations ordinarily being unavailable.

The records for group life insurance (that is, insurance issued to groups of employees generally with the cooperation of and contributions from the employer) have been used to calculate cancer death rates for all employees actively at work in particular industries. It should be noted that the cancer death rates for all employees actively at work in an industry so covered may not be significantly higher than average. Yet there may be some processes in that industry in which relatively small numbers of men are employed and which are associated with very high cancer death rates. However, it is frequently possible to determine cancer death rates for individual plants, and, if these plants concentrate on specific processes, the cancer death rate associated with such processes can be derived. A study covering the period 1927-1934 [17] indicated significantly higher than average mortality among felt hat makers, tube, rod, and pipe mill employees, paper manufacture employees, hotel and restaurant workers, shoe and light leather goods producers, and employees of electric and street railways.

In recent years a number of corporations have become interested in the mortality from cancer of specific sites among their employees, and such studies have been made in various industries or plants from group life insurance records. The customary procedure has been for the employer to approach the insurance company and request that

a study be made. Inasmuch as the relationship between the holder of a group life insurance contract and a life insurance company is confidential, a life insurance company is not free to publish the results for individual concerns. However, investigations made on an industry-wide basis are not subject to this restriction. At the present time, about 36 million persons are insured under group life insurance policies, and these include most of the employees of the larger companies in the major industries.

Industrial Insurance.—The records for industrial insurance (that is, insurance issued to individuals in small amounts on which premiums are payable weekly or monthly, usually collected at the home of the insured) can be used to compile cancer death rates according to the occupation of the insured at the time his policy was issued. It is usually also possible to ascertain the occupation of the policyholder at the time of his death. A large proportion of the adult males so insured are employed in industry. About 20 million adult males are currently insured under industrial policies, and these include, chiefly, the lower paid urban wage-earners.

From several studies made in the Metropolitan Life Insurance Company (based on the occupation at death), it would appear that the following occupations have been associated with higher-than-average death rates from lung cancer: coppersmiths and tinsmiths, painters and varnishers, and roofers and slaters. Less definite, but suggestive, have been the findings for such occupations as machinists and mechanics, railway enginemen and trainmen, proprietors and bartenders of drinking establishments, brick and stone masons, plumbers, steamfitters and gas fitters, and tailors and other clothing workers.

Special medicoactuarial records maintained by most of the larger life insurance companies on ordinary life insurance (that is, insurance issued to individuals in amounts of $1,000 or more) lend themselves most readily to the computation of cancer death rates according to occupation of the insured at the time the policy was issued. However, the last intercompany study of mortality among persons with ordinary life insurance according to occupation was made in 1937. This and earlier investigations indicated that cancer mortality was high among waiters and cooks, hotel keepers, guards, watchmen and janitors, painters and brick and stone masons, and tailors and clothing operatives. When studies of this kind are undertaken in the future, they will probably be made under the aegis of the Committee on Mortality under Ordinary Insurance and Annuities of the Society of Actuaries. About 60 million persons are currently insured under ordinary policies, comprising mostly middle-class males in nonhazardous occupations.

It is important to bear in mind that the records maintained by life insurance companies can merely indicate the fact that cancer mortality has been high in certain occupations. It does not by any means follow that such cancers are occupational in origin. Additional investigations must be made to determine whether the association is causal.

The records of accident and health insurance companies on operations and treatments for cancer provide another starting point for investigations of cancer mortality. However, in such instances an elaborate and costly machinery would be necessary to obtain a reasonably complete follow-up. Such a follow-up is automatically obtained where a person carries life insurance. The disability claim records of life insurance companies can be used as a means of determining the survival and recovery rates among persons insured under ordinary life insurance policies who submit disability claims on account of cancer. Such disability claim records usually contain more detailed information as to the site and type of cancer than do claim records. Furthermore, persons insured under ordinary life insurance policies who submit disability claims on account of cancer can, as a rule, be traced after recovery to determine the rate of recurrence of cancer.

The records of periodic health examinations of life insurance company employees have a potential value for the investigation of the natural development of cancer in its preclinical stages, even though employment by a life insurance company does not carry any known occupational cancer hazard.

Industrial Records

The use of public health and insurance company statistics in the study of occupational cancer can at best only indicate areas in which detailed studies should be undertaken. These must be undertaken within the industry itself and require the careful cooperation of several different groups. Since exposure within industry is far more likely to be greater than outside of industry, it is probable that studies conducted in the primary producers of a chemical will be more productive than studies of secondary users. Such investigations are difficult and time-consuming to conduct, and unless planning for them is undertaken in advance, they may be impossible to complete.

The reason for the lack of such planning in the past is found in the history of industrial medicine. At the turn of the century, industrial medicine was primarily concerned with the treatment of accidental injuries. This state of affairs still exists in many industrial plants, although the more progressive industrial physician is increasingly recognizing the importance of preventive and constructive health measures. Intensive safety campaigns and much safety engineering have reduced indus-

trial accidents to the point approaching the infinitesimal. Thus, only 3% or less of absenteeism in industry is due to in-plant accidents. The increasing importance of preventive-constructive measures in industrial medicine is attested by the recent recognition granted occupational medicine as a section of the American Board of Preventive Medicine.

Suspicion for the existence of an occupational cancer may also arise from the clinical judgment of the physician. Many medical advances have arisen out of the "clinical hunches" of well-trained and observant physicians. It is necessary only to point out that the clinical hunch can do no more than arouse suspicion; it neither proves nor disproves the existence of occupational cancer. However, when the clinical hunch serves as the basis of careful, detailed study, it may lead to significant advances in our knowledge. Such clinical hunches may arise out of a physician's being impressed by the number of cases he sees in a relatively restricted group, by corollary clinical findings in individual cases, or even by inquiries from the workers themselves, the answers to which he may not know but yet feel obligated to develop. The increasing potential significance of occupational cancer arising from the increased quantity and diversification of chemicals used in or made by industry places an increasing responsibility on the industrial physician, full or part-time.

When a suspicion has arisen, from studies of public health or insurance statistics as outlined above, by clinical judgment, or from an unusual location of a cancer, such as scrotum or bladder, that an occupational cancer may exist in a given industry, subsequent studies follow a pattern which it may be helpful to outline. The first step is to obtain all of the cancer cases of that industry for a period of time (perhaps 5 or 10 years) and to analyze them for over-all incidence in the industry. It is recognized immediately that the first requisite is that some system of cancer reporting within the industry is necessary. Let us assume, for example, that in a given industry 50 cases of cancer have been reported over the past 10 years. If this occurred in an industry in which there was an average annual employee census of 200, this would represent an incidence of 2,500 cases per 100,000 per year, which would be distinctly abnormal. If this incidence happened in only one year, it could well be a statistical artifact; hence, it is important to have the study encompass more than one year. On the other hand, if this occurred in an industry in which the average annual census was 2,000, this would represent an incidence of 250 per 100,000 per year, which would be just about the anticipated rate. If it happened in an industry with an average annual census of 20,000, this would represent an incidence of 25 per 100,000 per year. Such a result would clearly be so much below the expected that it should be immediately evident that only about 10% of the cases are actually being reported with the system used. Such a result should lead to a critical evaluation of the effectiveness of the reporting system. More detailed studies would be futile in this stage unless the cancer being studied is of an unusual site, such as the scrotum or bladder.

Let us assume, however, that an incidence of 250 per 100,000 per year has been determined. The next step would be to analyze the cases by sex. Thus, if it were found that 40 of the 50 cases occurred in men and that about 80% of the employees were male and 20% were female, the incidence rates of the sexes would be approximately the same. On the other hand, if 40 of the 50 cases occurred in men in an industry in which only 20% of the population were men, it would certainly be suggestive of two situations: first, that the incidence in men was suggestively high (i. e., 1,000 per 100,000 per year) and second, that the incidence in women inordinately low (i. e., 60 per 100,000 per year). Both situations would warrant further study in an attempt to understand the causes.

The next step in the procedure would be to pull out the medical record of each of the 50 cases reported and study it in great detail. The importance of an adequate medical record, containing a maximum of information and documented by originals or copies of laboratory reports, x-ray reports, biopsy reports, summaries of hospitalizations, autopsy records, and death certificates (when death has been the result), now becomes apparent. In looking to the future, every physician, and particularly every industrial physician, can understand the importance of the maintenance of adequate industrial medical records. It is apparent that if the industrial medical record is inadequate the promise of rewarding results from the detailed study of them is indeed remote.

However, after the medical records have been found adequate, detailed studies of them will now permit the proper classification of cases. For instance, such studies may reveal that cases reported as lung or liver cancer are, in reality, metastases of cancers of different primary sites. They should, therefore, be reclassified as to the primary site of origin. In addition, it may be found that certain conditions initially believed to be cancer and reported as such have been found on detailed study to be some disease other than cancer. The initial report on such cases may never have been changed and the cases carried erroneously as cancer cases. Such detailed study, therefore, may lead not only to an alteration of the total cases reported but also to changes in the distribution of cancer cases in organ systems.

This detailed study of the medical record also permits all cases to be classified by age at apparent onset of the disease. This study may be rewarding

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because if it is found that the average age at onset of cancer, either in general or in site-specific situations, is significantly lower than in the general population this itself may be justification for pursuing the studies further.

The next step in the procedure is to obtain an occupational history on the individuals with cancer. This is not an easy task, since the individual himself, if still living, may not remember all of the various occupations he has been involved in, even within one industry. At this point, the personnel and/or payroll office may be of great assistance to the investigator. Because it is necessary for industry to know the cost of its finished goods, cost accounting records are usually maintained. Since different jobs may demand different rates of pay, it is frequently possible by reviewing such records to follow a man's career within an industry in quite specific detail, at least as far as different departments and the major classifications within each of these departments are concerned. A study of such occupational records may then provide a further clue for the continuation of these studies. Thus, if an inordinate number of cancers appear among one group of workers or if cancers of a specific site all occur in men with a long service in one department, further detailed studies again may be warranted.

At this stage, the numerator of the incidence equation is available, but the denominator is not. This equation is as follows: Incidence (cases per 100,000 per year) = total number of cases for n years ÷ population at risk × n × 100,000. Let us assume that, at this point, examination of the records shows that there are 10 cases of lung cancer among gadget wreckers. Further review of the records discloses that among these 10 persons the minimum service as a gadget wrecker is nine years. Two more men have had 10 years of such service, one 11, two 13, one 15, one 18, one 23, and one 25 years. It would then be extremely helpful to the physician if he were able to obtain from the personnel department the number of men who have worked nine years or longer as a gadget wrecker. This figure would supply him with the denominator in the above equation. Thus, if our study encompasses 10 years, ending Dec. 31, 1955, and 100 men have worked nine years or more as a gadget wrecker by that date, the equation becomes as follows: Incidence = $10 \div 100 \times 10 \times 100{,}000 = 1{,}000$ per 100,000 per year. This incidence of lung cancer is some 30 times the expected incidence in men of all ages and about 14 times the expected incidence in men over 45 years of age.

It might appear that the study is now complete, but actually it should proceed further. The next step would be to obtain from the personnel department the names of all men who have ever, regardless of the duration of such work, worked as a gadget wrecker. Then the medical records of each of these men should be reviewed in order to insure that no cases of cancer have been overlooked. Experience in occupational cancer studies has shown that this review may frequently uncover proved cancer cases among these medical records, which, for one reason or another, may have escaped reporting. It is conceivable that such studies will reveal cases with an exposure time which is less than the previous minimum uncovered in the reported cases and, hence, may require revision of the population at risk. This type of analysis of medical records will insure that no cases have been missed. The collection of data on total employment in the department will permit analysis of cancer data in a variety of ways, most of which will give negative results which only serve to establish more firmly the significance of the positive results in the more selected groups. Thus, analysis of incidence rates for all men employed one day or longer, 5 years or longer, 9 years or longer, and 15 years or longer may show no statistically significant increased incidence until employment has exceeded 9 years, and an increasingly significant increase the more employment has extended beyond 9 years. It is obvious from what has been said that when the incidence rates have been determined they should be analyzed for statistical significance in comparison with the expected rates in the general population. This analysis should generally be performed by someone well acquainted with statistics as applied to biological problems. This is important because a four-fold or five-fold increase in incidence, which may appear on the surface to be significant, may really not have any significance statistically because of the small number of cases.

In addition, consideration should be given to the possibility that a difference in the characteristics of the industrial population and the general population with which it is compared may account for the observed difference in incidence. The incidence of cancer of specific sites is known to vary by age, sex, race, and geographical region, so that the possible effect of these factors should be investigated in the interpretation of differences in incidence rates.

Finally, when these rates have been determined, one last step may be worthwhile. This is to go to the foreman of the operation involved and inquire about the existence of shift books. A careful study of such shift books may reveal a pattern of occupation by subdivisions in the department which cannot be determined from the records maintained by the personnel and/or payroll office. It may be found that, although 100 men work in a department, perhaps only one-half or one-quarter of them are involved in one particular operation of the de-

partment and that all of the specific cancer cases are to be found among those men performing this specific operation. This final study may be of great importance from two standpoints: first, it may increase the significance of the incidence rates, and second, it may serve to pinpoint the exposures and, hence, lead to the easier detection of the carcinogenic agent and, thus, to its ultimate control.

It is necessary to point out that, in small industries with a heavy labor turnover and also in certain industries with problems peculiar to the industry, such as the construction industry, industrial medical records may be quite incomplete or even lacking. In such cases, obviously, the above techniques cannot be applied. Other methods, such as the use of social security records or high-caliber union medical services, may need to be developed for statistical, epidemiological studies of this nature. In these cases, again, the first requisite will be the collection and periodic analysis of adequate medical records.

From what has been said above, it can be seen that occupational cancer studies even within an industry can be laborious, time-consuming procedures. There are no real shortcuts to such adequate studies, and attempts to perform "quickie" or ill-conceived studies may lead to very misleading results. Adequate studies require the services of a physician who is interested in such studies and trained well enough to determine the adequacy of medical records. He must also have enough familiarity with the industry being studied in order that occupational histories will have some meaning to him. He must, in the course of such studies, enlist the support of the personnel and/or payroll departments, a competent biostatistician, the foreman, and other groups within the industry, including, particularly, engineers and chemists who are familiar with the operations being studied. Finally, he needs, for comparative purposes, age, sex, and site-specific incidence rates for the general population and for segments of the population more comparable to those he is studying, if his group differs in any significant way from the general population.

The above comments outline certain requirements for the future which will greatly assist in the performance of occupational cancer studies. First, and most important, are adequate industrial medical records. Second is a mechanism of recording and reporting disease which is capable of periodic analysis. This suggests McBee sort cards and IBM or other mechanical methods of analyzing data. Third is a mechanism of recording occupations and the occupational exposures of various segments of the working force. These occupational data must be capable of mechanical analysis so that a periodic running survey of occupational exposures, including their length, may be correlated with the medical data. If industry and industrial physicians become cognizant of the demands which will be made of them in the future and plan at this time for the recording of data so that they can be analyzed, it will be possible for them to become aware of any significant shifts in the incidence of disease, including occupational cancer, in the working force. The earlier such shifts are recognized, the sooner can their causes be studied and understood. This in turn will lead to the control of significant exposures and the genuine fruition of industrial medicine, whose confines lie within the preventive-constructive framework of medicine. These comments, although true of occupational cancer, are equally valid for all occupational disease. The methodology can be applied equally successfully by the industrial physician, the private physician, the public health physician, and the union physician, but, because of the need for assistance from other groups within industry, it will probably be most successfully applied by the industrial physician. It will become important for industry to know that its engineering controls are keeping pace with its technological developments, and this can most effectively be done through studies comparable to those outlined above.

Summary

In the majority of cases, occupational cancer cannot be distinguished, morphologically, from cancer not due to occupation. As a result, the diagnosis of occupational cancer is more difficult than the diagnosis of many other occupational diseases.

The suspicion for the existence of an occupational cancer hazard may arise from clinical observations, from public health statistics, or from the statistics available from insurance companies. The value and limitations of each of these must be realized. Failure to realize the limitations of these methods may lead to misleading conclusions. Failure to appreciate their value may result in overlooking important leads for further detailed studies.

In modern industry, with the tremendous expansion in the manufacture and use of chemicals, some of which may be carcinogenic, the responsibilities of the medical profession for detecting new occupational cancers have been increased. The first requisite in such studies is the maintenance of adequate medical records by competently trained physicians. In addition, it will be necessary to maintain adequate employment and occupational records. The correlation of cancer with occupation must be made from these records with a full appreciation of the natural history of nonoccupational cancer, such as its age, sex, and site distribution.

Specific increases in age, sex, and site-specific cancer incidence above normal incidences for the same age, sex, and site represent the only reliable

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evidence for the existence of an occupational cancer hazard. The importance of normal incidence rates in the general population, to serve as a baseline control, is therefore self-evident. Further, a careful methodology for occupational cancer studies from industrial medical and occupational records is required.

The important aspect of occupational cancer studies lies in the fact that an understanding of the disease will permit its control and, ultimately, its complete prevention. With modern engineering methods and control and protective devices, prevention can be achieved at reasonable cost.

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BLOOD SUGAR DETERMINATIONS.—Blood sugar in the majority of clinical laboratories is determined by obsolete analytical methods which yield false values. Instead of the actual glucose content of the blood, these values represent glucose equivalents of the combined copper reducing power of glucose plus other substances—such as glutathione, ergothioneine, uric acid, amino acids—which are entirely unrelated to blood sugar. The reducing power of these substances varies with the several reagents employed, so that the sugar values are distorted to variable degrees. Thus the non-sugar reducing matter adds to the actual sugar content 10-15 mg. percent in the Hagedorn-Jensen method, 15-25 mg. per cent in the original Folin-Wu method 25-35 mg. per cent by the old Shaffer-Hartmen method. In consequence, different laboratories report for the same sugar concentrations a variety of figures which allow no basis for comparison. This situation has been, and still is, the source of much confusion and misunderstandings. For 28 years now we have been using methods which give the true, correct sugar values. Since these are at least as simple as the methods which yield false values, there is no justification whatever for the inertia that keeps false values in circulation.—Michael Somogyi, Ph.D., Sugar Tolerance Tests: Their Relation to Diabetes and the Nature of Diabetes, Proceedings of the Forty-fifth Annual Meeting of the Medical Section of the American Life Convention in New York, 1957.

MEDICINE AT WORK

AT LAST—A HOSPITAL TO FIT DOCTOR-PATIENT NEEDS

Milton Golin

In Hartford, Conn., last month a middle-aged insurance executive checked into a hospital for an appendectomy. His doctor did a fine job and so did the hospital, which provided adequate nursing service, up-to-date facilities in the operating room and beside his bed, and prompt attention to his other needs. At the end of seven and a half days the patient paid a hospital bill of $229.20 and went home.

Good hospital care? The doctor had no specific complaint and neither did the patient—except perhaps to wonder how hospital costs have gone up in recent years. The fact of the matter is that for most of those seven and a half days the insurance man was exposed to care that was too good. He was paying for a great deal of institutional equipment and services that need not have been kept in readiness for him.

These included bedside oxygen and suction equipment which might have benefited a victim of a heart attack—but not him; a special bed with tucked-away pulley devices which would have helped a patient suffering from multiple fractures—but not him; and the continual availability of expert nursing surveillance which might have been a godsend to a patient with asthma—but not to him, at least not after the first day or two.

As things stand now in a large number of the 7,000 hospitals in the United States, almost every patient, no matter what his condition (except, perhaps, those in obstetric and pediatric units), has closely available to him the same equipment, the same facilities, and the same services as the next patient—and he has it throughout his hospital convalescence. Whether he underwent a critical operation yesterday or is sprucing up for departure in an hour, all the complex forces of the hospital are poised near at hand for his immediate service. Of course, if he is that asthma sufferer beginning to gasp for air, he may wonder how long it will take to summon a nurse from among those busy with patients who make relatively trivial demands.

How can this part-time waste of physical plant, specialized equipment, and expensively trained medical talent be reduced in order to give patients better hospital care for their money?

Pretty much the same question of cost and quality troubled Dr. Lowell T. Coggeshall in the spring of 1956 when he was appointed special assistant to the Secretary of Health, Education, and Welfare. Dr. Coggeshall (now dean of the University of Chicago's division of biological sciences) decided to launch a committee study to find some answers. The first launching brought much viewing with alarm and wringing of hands—but no accumulation of pertinent data which might lead to a solution.

Then Dr. Edward T. Thompson, already alerted to the problem, began putting some twos and twos together, in his recollection of the experiences of a few hospitals, and getting some interesting fours. This led to more memory-jogging and more study of some seemingly unorthodox but effective practices at several other hospitals.

As chief of intramural research activity for HEW's division of hospital and medical facilities, Dr. Thompson was in a position to direct his own Public Health Service study of the problem, rather than farm out the research with federal grants. Little by little he began uncovering evidence of a new trend in hospital care developing in the United States. He knew that, like a jigsaw puzzle, these pieces of evidence merely needed gathering and putting together to produce a full-dimensioned picture of what was happening.

How It Started

Dr. Thompson believes that the trend began developing shortly after World War II, when an increase in the number of major surgery cases led more and more hospitals to establish postanesthesia recovery rooms, where unconscious patients could be placed under intense and continual surveillance. (Of course many hospitals for years have had special units for the care of premature infants.)

"Soon," said Dr. Thompson, "internists and other physicians saw great benefits in these specialized facilities. They, too, arranged for intensive services to tide over patients stricken with critical heart, pulmonary and other conditions. At first, the hospitals restricted confinement of patients to only five or six specified hours of the day, usually the afternoon. When doctors expressed the need for evening use, too, a number of hospitals made intensive care facilities available around the clock on a short-term basis."

Thus began what is now being recognized by more and more physicians as "Progressive Patient Care"—the systematic classification of patients ac-

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cording to their medical needs. Dr. Thompson found only sketchy references to this system in hospital journals and other professional literature. It was largely a matter of poor communications—a hospital administrator in, say, Montana, not knowing about a hospital in Kentucky where patients were getting more effective service through an intensive care unit.

In February of 1957 Dr. Thompson decided to survey the extent of this trend. He located 20 hospitals which had intensive care units. He found others which lacked these units but did have special provision for patients to care for themselves during diagnostic examination, dietary regulation, daily physical therapy, and other treatment requiring minimum service of highly trained nurses.

In April of last year Dr. Thompson learned that Mr. Edward J. Thoms, administrator of the 180-bed Manchester Memorial Hospital in Manchester, Conn., was putting into action a bold plan which combined both these extremes of care into a three-phase program—gradually placing the patient in transition from special surveillance to self-help. (Or, the mobility might work in **reverse—from,** say, a diagnostic examination in the self-help unit, to surgery and the intense care unit, and then back to self-service by way of an intermediate care unit.) These divisions were representing broad areas of patient care—yet shading into one another to form a continuum of care based on medical need as determined solely by the patient's doctor.

Before long, Progressive Patient Care at Manchester was providing a higher level of service in a shorter period of time. Last July, encouraged by preliminary results, the Public Health Service decided to finance a detailed and continuing study of the Manchester experiment by a team of experts. There were to be no recommendations—just close scrutiny of the system in operation.

Meanwhile, the concept of Progressive Patient Care, abbreviated in medical circles to "PPC," was fast becoming known in scattered parts of the nation. By February of this year a tally of the number of hospitals handling patients in stages similar to the Manchester plan came to 30—a rise of 50% in a year. Last week the Public Health Service found nearly 150 hospitals in that degree of development.

"This whole idea is spreading rapidly," Dr. Thompson said last week in his Washington office. "Its ramifications are breath-taking. Many hospital administrators and practicing physicians see PPC as much more than a system of improved care. If it is accepted along proper guide lines by the hospitals of our country, a lot of other things could change for the better."

Changes Possible

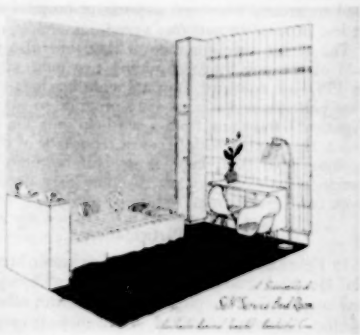
These are some of the wide-ranging beneficial changes he sees in the realm of possibility:

—Because only the doctor would decide when his patients were ready for transfer from one zoned unit to another, he would be brought into more intimate contact with the hospital than he is under most existing conditions.

—Installation of expensive equipment in only one section of the hospital, the intensive care unit, would bring about substantial reduction in over-all construction and operating costs. Design and architecture of the buildings also would be changed greatly.

—In turn, this prospect of accumulated savings might very well bring costs within the means of communities which now—in the midst of recession—are struggling with the problem of financing new hospitals or enlarging existing ones.

—Concentration of the most thoroughly trained nurses and ancillary medical personnel in the in-



Physicians stand by at an intensive care unit similar to the one under close study by a Public Health Service team. Scene at left is in Philadelphia General Hospital. At right is artist's conception of hotel room atmosphere of a self-service unit in Manchester (Conn.) Memorial Hospital, where the PHS study is centered.

tensive care units would allow more effective use of their skills. This is particularly important at a time of severe shortages in these fields.

—With more effective care, concepts of medical economics regarding so many hospital beds per so many thousands of population would be adjusted to more meaningful levels.

—Placing the patient in zones of medical need also would indicate the need for a new approach to prepayment hospital plans in which zoning costs might be covered—rather than through the present per diem subsidy according to socioeconomic standards by type of accommodation. In other words, policy holders might have the option of paying premiums for all three zones of hospital care, two zones, or one—with uncovered costs being paid out of pocket as a sort of deductible feature.

"And at last," Dr. Thompson adds, "there is the potential here for truly comprehensive internship and residency training to cover the combined experience of varied intensive care conditions in a single section of the hospital. Such an arrangement might expand or supplement the present systems of straight or rotating internship training in segregated medical, surgical, orthopedic, obstetric, and pediatric wards.

"Of course, these possibilities for progress represent great changes—changes which may be difficult to assess in the light of many decades of familiar hospital practices. Yet, over a period of time, they definitely could become realities from widespread acceptance of progressive patient care as the best kind of care."

Dr. Thompson is not alone in his optimism. Last December, shortly before details of the PPC plan were related to a House appropriations subcommittee, Dr. Leroy E. Burney, surgeon general of the Public Health Service, announced: "We hope this study at Manchester Memorial Hospital will lead to greatly improved patterns of hospital care at less expense to patients."

The House subcommittee at that time also was told of the possibility of adding two more stages to PPC—an adjoining hospital unit for long-term care patients, and a home-care program to smooth a patient's transition from a self-help unit. On this point, Dr. Burney said, "Tens of thousands of older persons have been kept in general hospitals for prolonged periods beyond the time they need or even can benefit from 'full-dress' services."

A. M. A. Participation

In February, as the study at Manchester Memorial Hospital was gathering steam, Dr. Thompson and members of the project team met with officials of the American Medical Association to report progress and preliminary findings. A. M. A. interest in PPC stems from the medical profession's continuing search for better total care for the patient.

In seeking the services of a medical advisory committee for the Manchester study, Dr. Thompson has had the help of (among other advisors) the A. M. A. Council on Medical Education and Hospitals and Council on Medical Service, the American Hospital Association, and the Joint Commission for the Accreditation of Hospitals.

Dr. Thompson reports that so far more than 5,300 hospitals have replied to a survey on intensive care and self-help units. Analysis of these returns is now being completed. Meanwhile, all 13 members of the project team at Manchester are completing their own evaluations of patient recovery there, acceptance of the new system, utilization of medical and nursing personnel, and costs to both patients and the hospital.

Late next month the combined findings will be presented to the surgeon general in the form of an initial report, "A One-Year Study at Manchester," along with a matrix of problems that require further investigation. The next step, says Dr. Thompson, will be publication of a guide for use by hospitals throughout the country, describing key problems in setting up Progressive Patient Care units, and including drawings of schematic models of the different types of units.

Already there is some solid evidence from Manchester that patients are not only getting better care under PPC but are experiencing a briefer hospital stay at less cost to them and to the hospital. According to comparative figures there, the middle-aged insurance executive cited earlier as the appendectomy patient in a nearby Hartford, Conn., hospital would probably have been released from Manchester Memorial in six and a half days (instead of seven and a half in Hartford) and would have paid a bill of $183.88 (a saving of $45.32). The same comparative data show that the Manchester hospital, under its PPC system, can figure an inventory of no more than $337 per bed, compared to an average inventory cost of $353 per bed in Connecticut hospitals offering a conventional single-phase system of patient care.

Varying Room Rates

One reason for the saving to patients is varying room rates. The scale at Manchester Memorial Hospital for accommodation in an intensive care unit (with continual nursing surveillance of four beds) is $22 a day. The rate drops to $16 when the patient is moved to an intermediate care unit, and to $8 a day in a self-service unit—where service by registered nurses is at a minimum, the patient is able to care for his own toilet, and he goes to the hospital cafeteria for his meals.

"While individual costs may be less under PPC," says Dr. Thompson, "this of course is not the system's most significant feature. The really big advantage lies in much better care for the patient's hospital dollar."

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While Dr. Thompson oversees the Manchester study from his Washington office, the field project in Connecticut is being led by Dr. Faye G. Abdellah, who is on detail from the division of nursing resources of the Public Health Service. Others on the 13-member team include a biostatistician, physicians, nurses, a cost analyst, a dietitian, an architect, an equipment analyst, an operations research specialist, a medical records librarian (Mrs. Adaline C. Hayden of the A. M. A.), and a social scientist and psychologist (who gauge the system's acceptance by patients, their families, and the Manchester community).

The work of this group might very well set off a chain reaction of improved hospital care—benefiting the more than one million patients who are in the hospitals of our nation every day of the year. To what degree PPC will influence planning for new hospitals and for the expansion of existing ones is impossible to gauge at this time. For all its promise, for all its status as a leading exponent in a decidely growing trend, PPC at Manchester still is not really too far beyond the experimental stage. Even under the best guide lines, acceptance of any radical concept—no matter how much improvement it indicates—can be a painful process.

Yet, in this period combining great cost-consciousness with great health-consciousness, even the preliminary evidence of PPC's established success in Manchester might provide impetus for the hospital expansion which our nation so sorely needs.

For example, two months ago—well before favorable results of the Manchester project were made known—the Hospital Planning Council for Metropolitan Chicago was created with the objective of meeting hospital needs more economically and efficiently than in the past. This already is being done at Manchester. The Council announced it would try to solve the problem of how "duplication of expensive resources can be minimized and how old hospitals can be adapted for future use." This also is being done at Manchester.

Other objectives of the Chicago council are to care for the sick "in accordance with the measured needs for these services" and to "provide a means whereby the interests of the hospitals and the medical profession may be more closely correlated." The fact that PPC is meeting these objectives, too, along with the others, is really no coincidence. Chicago's search for solution of its hospital problem is typical of the plight of scores of communities throughout the United States. It just happens that the broad problem is being crystallized for possible solution in Manchester.

In gauging whether the study at Manchester will set a national pattern, it is too early to suppose that probability will overtake possibility. But it is not too early to predict that the experience there, after being relayed as an official report to the Public Health Service in a few weeks, will evoke serious consideration of the organization and re-alignment of function of the 7,000 hospitals in the United States.

ANTIBIOTIC-RESISTANT STAPHYLOCOCCI.—Although hardly comparable with respect to numbers of organisms killed and the speed of killing, erythromycin and chloramphenicol in combined large dosage have proved to be an effective combination to control growth of penicillin-resistant organisms. These two, with perhaps bacitracin for a few days, have proved to be as effective a combination in therapy of human infections as has been described. . . . It has been our impression, both in vitro and in vivo, that staphylococci whose resistance to penicillin is greater than 0.15 u/ml. produce penicillinase and are for practical purposes, penicillin-resistant. Penicillin in clinical therapy is useless in infections caused by those strains of staphylococci. Similarly, other single-drug therapy has resulted in increasing numbers of strains of staphylococci resistant to multiple other agents. In such circumstances the agent is of no clinical value either. Irrespective of the importance of the epidemiologic aspects of penicillin-resistant staphylococci, it cannot be concluded that the infection hits only old persons in the hospital whose admission was necessitated by some other debilitating, noninfectious disease. . . . They do, however, probably occur more often in patients receiving antibiotics for some nonspecific "prophylactic" reasons than in patients not so "protected." To date, no agent or combination of them is as efficacious as pencillin was in the era when the majority of isolated strains were sensitive to it.—Paul Bunn, M.D., Aldona Baltch, M.D., Winifred Osborne, B.S., and Leonard Canarili, B.S., Staphylococci: On the Obiquitous Nature of Human Infections and Their Control by Antimicrobial Agents, Singly or in Combination, *Annals of Internal Medicine*, January, 1958.

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WHY DO FEMALES LIVE LONGER?

IT IS difficult to explain sex differences in mortality and longevity. These differences are being widely publicized as expectation of life at birth in the United States approaches three score years and ten. Moreover, the United States population census of 1950 was the first one to reveal more females than males —a surplus now approaching 2 million. This surplus of women is more pronounced among the higher age groups. In fact, some demographers point to the prospect of "a nation of old ladies." Lower mortality among females increasingly offsets and counterbalances the excess of male births.

Expectation of life at birth is about 73 years for white females and only 67 years for white males; in 1940 the excess for females was only four years. Expectation of life at birth is a severely qualified forecast. Currently, 1,000 white girl babies born during the year would be expected to live a total of 73,000 years if their mortality during each successive year of life were the same as now prevails. Few demographers would care to predict that age-specific mortality rates will remain unchanged, even in the 21st century, until all of these 1,000 babies have died. Likewise, the statement that 1,000 white male babies can be expected to live a total of 67,000 years, or an average of 67 years, is based upon the continuation of current age-specific mortality rates during their entire lifetime.

Whether this sex differential in longevity increases or decreases or will be greater or smaller in 1975 than it was in 1950 is a matter for speculation. Explanations of the current difference of six years are far from complete. Natural scientists are forever reminding us that the female outlives the male in most species of the animal kingdom where the question of longevity has been carefully studied. Longer life of women has been attributed to lesser pressures of living. Whereas a man has to work under greater strains in a highly complex civilization, women are aided in the release from household duties by all manner of appliances. The mortality risks of childbearing are very minor today, as attested by the precipitous decline in maternal mortality rates. Women can devote more of their time to leisurely living, especially as their children grow older.

Two sociologists, Professor Vance and Father Madigan,[1] call this social explanation of sex mortality differentials sex differences in the "style of life." They tested this by comparing the mortality of American Catholic religious brothers and sisters, who face somewhat similar occupational hazards, do not have families, and in general are subject to somewhat similar pressures of living; that is, the differences between the sexes in the "style of life" were considered minor. These two investigators reasoned that, if they found no sex differentials in mortality in this control group, the sex variations in the "style of life" could largely explain the lower mortality enjoyed by females generally in the United States. But the nuns (sisters) had lower mortality and lived longer than their male counterparts (brothers)! Expectation of life at birth in this control group was four years, or 10%, greater for the females than for the males. Since this excess of 10% in length of life for females was matched by a similar difference of 10% in favor of white females generally in the United States, the investigators concluded that "style of life" was not an explanation of lower mortality or longer life among females. Apparently the scientific explanations for longer life among females must be biological rather than social.

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MENTAL HEALTH WEEK

The 10th annual observance of mental health week will be held this year from April 27 to May 4. The co-sponsors are the National Association for Mental Health and the National Institute of Mental Health. The theme for the 1958 observance is, "With Your Help, the Mentally Ill Can Come Back." Emphasis is being placed in four major areas: (1) improved care and treatment for patients in mental hospitals; (2) expanded services for the early detection and treatment of mental and emotional illness; (3) more adequate rehabilitation services for the improved or recovered mental patient; and (4) expanded research into the causes, prevention, and treatment of mental illness.

Some gains have been made during the past few years. New methods of treatment, including the use of new drugs, are restoring more mentally ill pa-

tients to health. The stigma surrounding mental illness has been reduced. Additional research is getting underway, although for each patient being treated for mental illness we are still spending less than $4 per year on research to find new ways of prevention, treatment, and rehabilitation. Communities are providing more facilities for diagnosis and treatment of the mentally ill.

These and other gains are small in comparison to the unmet needs of the mentally ill. Mental illness remains the number one health problem of the nation. Over one-half of all the hospital beds in the United States are occupied by persons suffering from mental illness or disability. In addition to the more than 750,000 persons hospitalized because of mental disorders it is estimated that 16 million persons suffer from some form of mental or emotional disorder and are in need of psychiatric care.

Most of the hospitalized mentally ill are cared for in the state hospitals of the nation. These hospitals are for the most part overcrowded and understaffed to such an extent that many thousands of patients are doomed to chronic and often hopeless invalidism.

The average per diem cost per patient in the state hospitals of the United States for 1956 was $3.27. In 1957 this had increased to $3.64. The state with the lowest per diem cost per patient spent $1.85, the state with the highest spent $5.47. The District of Columbia spent $5.75, while the cost per patient per day in Veterans Administration neuropsychiatric hospitals was $10.31.

The average expenditure of $3.64 per day per patient in the state hospitals and the substandard care given constitute a national disgrace. The physicians of the nation need to be made aware of the enormity of the problem and stimulated to assume their responsibility to see to it that the patients in these hospitals receive the type of treatment and care to which they are entitled. Physicians not only have the responsibility to do this but are in a unique position to provide effective leadership to an increasing number of citizens and citizen organizations that are becoming aware of the problems and are mobilizing for corrective action.

A more hopeful aspect of the mental illness problem is the increasing interest of physicians and hospital administrators in providing psychiatric services as part of the general hospital care program, as well as maintaining private mental hospitals. In 1956 there were 209 private hospitals and 393 psychiatric services in general hospitals in the United States. As these facilities are increased more patients will be given prompt and intensive therapy and for most of whom the prognosis will be early restoration to mental health.

The American Medical Association played a vital role in the establishment of the Joint Commission on Mental Illness and Health and is continuing to assist in its work. It is hoped that this commission will soon make available to all legislative bodies and other interested groups outlines of those patterns of patient care most likely to restore the mentally ill to good health. Even more important, this commission may discover new principles of mental health and formulate them with those already known, to the end that the incidence of mental illness can be significantly reduced.

An opportunity for great service is presented to all physicians to help in the observance of mental health week.

NEW ERA IN HOSPITAL CARE?

Since April of 1957 the Manchester (Conn.) Memorial Hospital has been serving patients and physicians in an entirely new approach to institutional therapy, called Progressive Patient Care. It is a comprehensive system designed to adapt hospital facilities to the patient, rather than the other way around. As a Medicine at Work article points out on page 2180, the Manchester concept of care (refined, actually, from the experiences of a number of other hospitals) may foreshadow new thinking, new planning, and new policies in many of the 7,000 hospitals of the United States. Events at Manchester seem to impinge upon nonmedical as well as medical phases of the vast hospital field whose physical assets are estimated at 13 billion dollars. These phases include economics, internship and residency training, community relations, nurse and other ancillary personnel supply, architecture, doctor-hospital relationships, and health insurance plans.

HIGH-PRESSURE TELEPHONE SECURITIES SALESMEN

An increasing number of complaints against high-pressure telephone security salesmen has been received by the regional office of the U. S. Securities and Exchange Commission in New York City, and among the victims of their nefarious conduct has been a considerable number of physicians. The New York state attorney general and the Securities and Exchange Commission some time ago published a 10-point guide to alert the public against fraudulent securities practices, but the number of complaints still mounts. The commission has forwarded a letter and the guide to THE JOURNAL (page 2213) in the hope that members of the medical profession thus will be enabled to avoid the pitfalls involved in the purchase of securities from unknown sources. The 10-point guide is stated simply and clearly, and the investing physician will find it useful.

THE PRESIDENT'S PAGE

A MONTHLY MESSAGE

"The most important field of medical service in the future undoubtedly will be among the older people." I made this observation at the Governor's Conference on Medical and Hospital Services in Trenton, N. J., a little more than a year ago.

It is pleasing, therefore, to tell you that this month the American Medical Association has joined with the American Dental Association, the American Hospital Association, and the American Nursing Home Association to form the Joint Council to Improve the Health Care of the Aged. Every effort will be made to provide the best possible health care for America's older citizens and to aid in the solution of their various health problems.

I personally am delighted to be one of A. M. A.'s three representatives, along with Drs. Gunnar Gundersen, A. M. A. President-elect, and F. J. L. Blasingame, A. M. A. General Manager, on this potentially history-making joint council. We promise to uphold its objectives: "To identify and analyze the health needs of the aged, to appraise the available health resources for the aged, and to develop positive health care programs regardless of the economic status of the aged."

Your A. M. A. also pledges its full resources, including manpower and idea power, in working through the joint council for the health and well-being of the present 14 to 15 million persons in the over-65 age group and the millions who will come into this bracket in future decades.

Because our profession has been largely responsible for constantly increasing the life span of man in the United States, it is only fitting that medical doctors do all within their power to develop programs and facilities tailored to meet the health needs and finances of the aged.

The A. M. A.'s Committee on Aging and the Council on Medical Service have provided active and aggressive leadership in the field of health care for the aged. Your A. M. A. recognizes, however, that the solution of the problems of the aging for the present and future demands joint planning by medicine and other major providers of health care to the aged. Therefore, the joint council has been established to coordinate the efforts of the participating associations, to intensify the research of each sponsoring group, and to activate projects as rapidly as possible.

Individual members of the joint council already have undertaken special studies of the economic assets and resources of the aged, the extent and adequacy of voluntary health insurance for the over-65-year group, the utilization by the aged of hospital and nursing home facilities, and the review of all health programs of special value to the aged.

In addition, the joint council is reviewing existing facilities with special reference to the aged, particularly nursing home facilities with the hope of improving and increasing them. Another facet of the joint council's broad-range program will be to work closely with health insurance groups in an effort to improve the coverage of the aged and to see that their insurance dollars go further.

The joint council also believes that much can be done for older persons by the states and communities and it hopes to promote increased financial support for medical, hospital, and nursing home care of the aged from these local sources for those who cannot afford to pay.

It is impossible to give you a complete picture of the proposed program of the joint council, but I believe you get some idea of the magnitude of the endeavor and the unlimited opportunities for service in this field.

Medicine always has given more than its share to America and Americans. In coping with the problems of the aged, medical doctors once again are called upon for the best in the personality of medicine. I have the fullest confidence that our profession will deliberate intelligently, plan wisely, and act vigorously in the interest of our senior citizens.

David B. Allman, M.D.
Atlantic City, N. J.

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ORGANIZATION SECTION

A. M. A. APPROVAL OF S. 1045

March 25, 1958

Honorable Warren G. Magnuson, Chairman
Senate Committee on Interstate and Foreign Commerce
United States Senate
Washington 25, D. C.

Dear Senator Magnuson:

On April 2, 1957, a letter was sent to you by the American Medical Association stating our reasons for not testifying at that time on S. 1045, 85th Congress, a bill to establish an Office of Civil Aviation Medicine in the Civil Aeronautics Administration. We advised you that our Association was reviewing material in this field, and until its completion we would not be in a position to offer testimony on the bill. We requested you to defer action until we could supply your Committee with the results of our study and the recommendations resulting therefrom.

Our survey was initiated by an *ad hoc* committee studying the organization and functions of civil aviation medicine. A permanent Committee on Aviation Medicine has now been appointed by our Board of Trustees to continue the inquiry. On that Committee's recommendation the A. M. A. has adopted a position of active approval of S. 1045.

The American Medical Association is seriously concerned with the present state of civil aviation insofar as medical matters are concerned. Increasing air traffic and the jet age, accompanied by increased physical demand on pilots require a substantial revision in the present method of handling many aspects of aviation medicine. Specifically, a separate medical office should be established in the Civil Aeronautics Administration with the administrative status of the Office of Civil Aviation as proposed in S. 1045.

The establishment of physical standards for and the medical evaluation of civil airmen must be carried out by physicians experienced in aviation medicine. Because of the operational tasks of airmen, their physical and mental fitness, and ability to perform under normal and emergency conditions must be assured. Medical determinations should be conducted by personnel who have the necessary professional qualifications and experience.

The proposed Civil Aeronautics Medical Research Laboratory seems desirable and necessary.

We approve the "Office of Civil Air Surgeon" proposed in S. 1045. We feel that he should be directly responsible to the Civil Aeronautics Administrator in matters of policy, administration, regulations, etc. We understand that the direct responsibility of the Civil Air Surgeon to the Administrator is not clear at the present time to some persons. We would suggest that steps be taken to clarify this relationship.

We endorse the transfer of the rule-making authority for medical standards from the Civil Aeronautics Board to the Civil Aeronautics Administration. These government regulations should be developed, drafted, promulgated and administered by competent and qualified specialists. Unlike the CAA, the Civil Aeronautics Board does not have qualified medical personnel.

While we do not suggest that this bill is the only method by which the needed reforms in civil aviation medicine may be accomplished, and while we do not know whether the administrative organization proposed by this bill is the best possible one in all details, we do believe that S. 1045 represents the best mechanism currently proposed for accomplishing necessary reforms. (In December, 1957, the House of Delegates of the American Medical Association adopted a resolution on this subject.) Although a copy of this resolution has already been supplied to you, I am attaching another copy for the information of your Committee and for inclusion in the record.

Sincerely yours,
F. J. L. Blasingame, M.D.
General Manager

• NEW A. M. A. PUBLICATIONS

An A. M. A. pamphlet, "Are You Fit to Drive?" urges drivers to contact their physicians if they are in doubt about their fitness. Prepared by the A. M. A. Committee on Medical Aspects of Automobile Injuries and Deaths in cooperation with the Center for Safety Education at New York University, the booklet contains information about those conditions that can adversely affect driving skills—emotional upsets, driver attitudes, sleepiness, medicines, faulty vision, certain nerve and heart disorders, diabetes, old age and drinking. For distribution through physicians' offices, the booklet is available from the Association of Casualty and Surety Companies, 60 John St., New York 38. Price is $4.60 per 100 copies, regardless of quantity.

A new A. M. A. pamphlet entitled "Protecting the Health of the High School Athlete" calls for the

sponsoring of high school sports injury conferences to instruct coaches, athletic directors, and team physicians on the early recognition of injuries, appropriate first-aid measures, and the prompt referral of injured players for medical or dental care. The booklet was prepared under the auspices of the A. M. A. Committee on Injury in Sports. Copies may be secured from the A. M. A. Bureau of Health Education.

FEDERAL MEDICAL LEGISLATION

Second Session, 85th Congress

Social Security

S. 3086, by Senator Proxmire (D., Wis.), is of vital importance to the medical profession in that it is similar to the Forand Bill H. R. 9467 as it provides, among other things, a program of free hospitalization. To some extent, however, it goes even further than does the Forand Bill. The bill would provide a program of free hospitalization of 60 days in any 12-month period for (*a*) persons eligible for retirement benefits; (*b*) dependents of persons eligible for retirement benefits; and (*c*) dependents of individuals eligible for disability benefits (but not the disabled individual himself). The secretary of the Department of Health, Education, and Welfare would enter into agreement with individual hospitals to provide hospital services, defined as drugs and appliances furnished by a hospital to any individual as a bed patient: bed and board and such nursing service, laboratory services, ambulance services, operating room, staff, and other services, and drugs and appliances as are customarily furnished by such hospitals to its bed patients, either through its own employees or through persons with whom it has made arrangements for such services, drugs, or appliances. This includes such medical care as is generally furnished by hospitals as part of hospital care for hospital patients. Such terms shall not include care in the tuberculosis or mental hospital.

The secretary would be authorized to utilize the services of private nonprofit organizations representing "qualified providers of hospital services" or which "operate voluntary insurance plans" to reimburse hospitals for service.

The bill would increase the wage base upon which Social Security taxes are paid from the present rate of $4,200 to $7,500, beginning Jan. 1, 1959. Maximum monthly family benefits would increase from the present rate of $200 to $334. The bill would provide a new formula which would increase the benefits paid to an individual. It would also amend the Internal Revenue code to make the $7,500 wage base subject to an increasing tax rate to the extent that, beginning in 1975, the self-employed individual, with an income of $7,500 or more, would pay $534.38 for Social Security coverage; the employed individual and his employer would pay $375.00 each. These are only a few of the highlights of the bill by Senator Proxmire.

H. R. 10532, introduced by Representative Dellay (D., N. J.), is identical with the Forand Bill, H. R. 9467, to amend the Social Security Act to increase benefits under the Old Age, Survivors, and Disability Insurance Programs, and to provide insurance against hospital and surgical services for persons eligible for retirement benefits. The import of the Forand Bill is found in an editorial in THE JOURNAL, (165:1568 [Nov. 23] 1957) and in the President's Page (*J. A. M. A.* 165:1701 [Nov. 30] 1957).

H. R. 10578, introduced by Representative Dingell (D., Mich.), is similar to S. 1811 by Senator Revercomb (R., W. Va.), and would redefine disability for purposes of entitlement to disability insurance benefits and the disability freeze. The applicant, who meets other requirements and could not obtain employment in any occupation similar to the work he engaged in prior to his disability because of his impairment, would be eligible for disability payments or freeze benefits.

H. R. 10583, introduced by Representative Dingell (D., Mich.), would amend the Social Security Act to provide that certain determinations of disability made by other federal or state agencies shall be accepted as proof of disability under the Social Security Act. More comprehensive analysis of the bills mentioned can be secured if desired by contacting the Washington office of the A. M. A.

Welfare and Benefit Plans

Congress was apparently impressed last year at the hearings by the evidence of mishandling of funds of union-sponsored welfare and benefit plans by union officials to the extent that the number of bills to place such plans under federal regulation and control continues to grow. One of the latest bills to be introduced is H. R. 10102 by Representative Berry (R., S. D.), which is identical to S. 3044, by Senator Mundt. H. R. 10236, by Representative Holt (R., Calif.), and H. R. 10272, by Frelinghuysen (R., N. J.), are identical to that of S. 3097, by Senator Smith (R., N. J.), which is the administration's bill. It is identified as the "Labor Reports Act of 1958" and would require labor organizations to file, with the Secretary of Labor, information as to their organizational structure, officers, activities, procedures, and financial activities, including welfare and benefit plans. Employers would also be urged to file reports on transactions with labor organizations, including participation in welfare benefit plans. An office of Commissioner of Labor Reports would also be created in the Department of Labor and would carry out the intent of the bill and administer the regulations established by the Secretary of Labor. The secretary would

From the Washington Office of the American Medical Association.

have subpoena power to require the submission of reports and financial data. A labor union which willfully failed to make the reports required by the bill or to comply with any regulations established under it would (*a*) lose its rights and privileges under present law; (*b*) not be permitted to appeal to the National Labor Relations Board for aid and relief; and (*c*) lose its tax exempt status. An employer failing to submit the required reports would not be able to appeal to the NLRB for relief. Individuals failing to comply with the provisions of the bill would be subject to a fine up to $5,000 and/or five years in jail.

Senator Knowland (R., Calif.) introduced S. 3068 for the purpose of insuring democratic control of labor organizations by membership. To accomplish this, the bill would amend the Labor Management Relations Act of 1947 to provide referendums by secret ballot if requested by 15% of the membership of a union for the purpose of (*a*) amending, modifying, or revising the organizational structure or procedures of a union; (*b*) election or re-call of officers; and (*c*) the protection of members from unfair labor practice. An "employee welfare plan" is defined as a fund or program established by the employer providing benefits in the case of illness or disability or upon retirement or death of the employee. These welfare plans would be registered annually with the Securities and Exchange Commission. The registration would contain information as to (*a*) the nature and type of plans and the benefits provided under them; (*b*) number of employees covered; (*c*) assets and investments of the funds; (*d*) receipts and disbursements during the proceeding year; and (*e*) whatever other information the commission might require.

If violations of the law were found on examination of the report by the commission, the attorney general or appropriate state enforcement agency would be informed. The commission would then have subpoena power to enforce its requests for information about their plans. Officers failing to regulate welfare plans as required by the commission would be liable to fines up to $1,000 or one year in prison or both.

Tax Department

New bills to liberalize tax deductions for various expenses continue to be introduced. In regard to medical expenses, Representative Matthews (D., Fla.) introduced H. R. 10394 which is identical to H. R. 7347 by Representative Dellay (R., N. J.) and which would eliminate present provisions that restrict medical expense deductions to the excess of 3% of taxpayer's adjusted gross income. It would allow as a deduction all medical expenses not covered by insurance, including drugs for a taxpayer or his dependents. S. 3226, introduced by Senator Long (D., La.), is identical to H. R. 9633 by Representative Boggs (D., La.) and to H. R. 9417 by Representative Miller (R., Md.), and would permit a taxpayer 65 years of age or older, who is disabled, to deduct medical expenses for himself and for his spouse if age 65 and also disabled, not to exceed $20,000 in any one year.

Tax deduction for higher education is the subject of H. R. 10506 by Representative Scott (R., Pa.). H. R. 10543 by Representative May (R., Conn.) and S. 3162 by Senator Smathers (D., Fla.) are similar to the many other bills mentioned on tax deduction for higher education. The main difference in the bills is in the amount of deduction to be allowed for higher education, and whether or not the deduction would effect an increase in the personal income tax exemption or would be taken as a straight deduction. H. R. 10506 would increase the personal tax exemption from $600 to $1,000 for a dependent attending college. H. R. 10543 would allow a deduction of $800 per year for tuition and fees for a taxpayer or his spouse or his dependents if they are students for five calendar months at an educational institution above the 12th grade. S. 3162 would allow the taxpayer to deduct all expenses directly attributable to attendance at college for himself, his spouse, or his dependents.

Something new and of special interest to physicians are the following bills identical to each other: H. R. 10617 introduced by Representative Judd (R., Minn.), H. R. 10499 introduced by Representative Rains (D., Ala.), and S. 3194 introduced by Senator Sparkman (S. 3194 now has 34 senators as co-sponsors). These bills would amend the Internal Revenue Code of 1954 so as to establish an initial program of tax adjustment for small and independent businesses and for persons engaged in such businesses. Also included is a Jenkins-Keogh type tax deferment proposal. This would permit any individual who is not covered by or receiving payments from an employer's pension, annuity, or stock bonus plan to deduct from his taxable income an amount paid by him each year to obtain retirement income for himself or payments to his beneficiaries. The annual payments, called "retirement deposits," would be limited to 10% of the adjusted gross income or $1,000, whichever is less.

For persons between ages of 50 and 70, an additional 10% of the above formula could be added for each year over the age of 50. If an individual did not make retirement deposits equal to the amount allowed him for any given year, he could increase his deductions over the next five years by an amount equal to, but not exceeding, the total of his "unused deduction." Retirement deposits could be in the form of money placed in a restricted retirement fund operated by a bank, or as premiums under a restricted retirement policy issued by a life insurance company.

Periodic payments made to the taxpayer after age of 65 or to his beneficiaries would be treated as ordinary income for tax purposes. If a lump sum distribution is made to the taxpayer after he has reached the age of 65, and if the plan or policy has been in effect for five years, the tax would be no more than five times greater than the increase resulting if only 20% of the total has been distributed. The same tax rate would be applied if the lump sum payments were made to the taxpayer's estate or his beneficiaries after his death.

Veterans

The bill by Representative Weaver (R., Neb.), H. R. 10130, identical to H. R. 10028 by Representative Teague (D., Texas) provides that no VA hospital, domiciliary, medical center, or regional office could be closed or transferred unless the House Veterans Affairs Committee (of which Representative Teague is chairman) is notified 90 days in advance. H. R. 10134 by Representative Winstead (D., Miss.) is identical to H. R. 9726 by Representative Smith (D., Miss.) in that it provides that the VA shall not sever service connection for any veteran's disability when said veteran has been in receipt of compensation for 10 or more years.

Surplus Property

The bill by Senator Holland, S. 3121; H. R. 10010 by Representative Herlong; and H. R. 10118 by Representative Matthews (Democrats, Fla.), are identical bills and deal with authority to dispose of certain federal surplus property to public health agencies and states. "Public Health" is defined to include sanitation activities, such as sanitary land fill programs, mosquito and insect control programs, malaria control programs, and drainage programs. Also included in the definition of "public health agencies of the state," are those agencies carrying on the above programs.

Miscellaneous

H. R. 10177 introduced by Representative Lane (D., Mass.) would create a presumption that certain impairments of health caused by hypertension or heart disease of a federal or District of Columbia employee is incurred in line of duty for purposes of certain retirement and disability compensation laws or system.

H. R. 10178 introduced by Representative McDonough (R., Calif.) would authorize 12 million dollars for a six-year period, (two million dollars yearly beginning with fiscal year 1958) for grants to states and public and nonprofit organizations for establishing methods and techniques for understanding the magnitude, characteristics, and needs of the physically handicapped and for developing methods of coordinating available resources to increase the activity of handicapped individuals. Data collected as a result of these studies would be used to project the needs and to determine the federal government's future role in the vocational rehabilitation field.

H. R. 10310 introduced by Representative Anfuso (D., N. Y.), and S. 3167 by Senator Javits-Ives (R., N. Y.), are identical and would provide for the construction of a public health hospital in the state of New York to be used in the treatment of drug addicts, and would possibly be located on Ellis Island.

H. R. 10404 introduced by Representative Williams (D., Miss.) is similar to H. R. 6747 by Representative Harris (D., Ark.), and S. 1895 by Senator Hill (D., Ala.) and would prohibit the use of new food additives which have not been adequately protected to establish their safe use under the conditions of their intended use. "New food additives" is defined in the bill as "any substance . . . intended to be used . . . in the manufacture, packing, processing, preparation, or other fabrication of any food and thus to become a component of such food, but which is not generally recognized, among experts qualified by scientific training and experience . . . as safe for use under the conditions of its intended use."

S. 3089 by Senator McNamara (D., Mich.) and Senator Humphrey (D., Minn.) is identical to H. R. 9822 by Representative Fogarty (D., R. I.) and S. 2994 by Senator Newberger (D., Ore.) and Senator Kefauver (D., Tenn.) would provide for a White House Conference on Aging and to assist the states conducting similar conferences on aging prior to the White House Conference and prior to Dec. 31, 1958.

REFERRAL CENTERS FOR CHRONICALLY ILL

A survey of five information and referral centers for the chronically ill is being conducted by the A. M. A. Council on Medical Service. Located in Chicago, Cleveland, Milwaukee, San Francisco, and Essex County, N. J., these centers operate as clearing houses for information on facilities for the care of chronically ill in the community. The Council's report on these centers should be of interest to physicians concerned with chronically ill patients. Any information on similar centers in other communities will be welcomed by the Council.

JOURNAL INDEX

The index to volume 166 of THE JOURNAL will appear in the May 10, 1958, issue. Those who wish extra copies of the index may receive them, without charge, on request to the Order Department, American Medical Association, 535 N. Dearborn St., Chicago 10.

COUNCIL ON MEDICAL EDUCATION AND HOSPITALS

POSTGRADUATE COURSES

The following notices of postgraduate courses for physicians have not previously been published by the Council and are presented for information only:

An intensive three-day, 15¼-hour course, **Clinical Electrocardiography,** is scheduled May 2-4, 1958, at the Huron Road Hospital, 13951 Terrace Rd., Cleveland 12, for physicians in general practice. Methods of education will be lectures with visual aids, concluding with interpretations of "unknown" tracings by the panel and the audience. The fee will be $25. Additional information may be obtained by addressing the Staff Office of the hospital.

An intensive, multidiscipline course, **Epidemiology and Control of Food-Borne Diseases,** will be offered May 19-23, 1958, at the Communicable Disease Center, U. S. Public Health Service, 50 Seventh St. N.E., Atlanta 23, Georgia. It is designed to provide physicians, veterinarians, nurses, and other members of public health teams with current information and techniques on the subject. Further information may be obtained from Chief, Training Branch, at the Center.

The first annual postgraduate **Seminar in Pediatrics** at the Children's Hospital, 2125 Thirteenth St. N.W., Washington 9, D. C., will be conducted by the faculties of Georgetown and George Washington Medical Schools in cooperation with the National Institutes of Health and the military medical centers of Washington May 22-24, 1958. The fee will be $25. Further information may be obtained from the Director of Medical Education of the Children's Hospital.

A three-week intensive course on **Tuberculosis and other Pulmonary Diseases** is scheduled for June 2-20, 1958, at the Trudeau Foundation, Saranac Lake, N. Y., at a fee of $100. Enrollment will be limited. Inquiries may be addressed to Box 500, Saranac Lake, N. Y.

An intensive five-day course, four hours each afternoon, on **Normal Ocular Histology and Histopathology** will take place June 2-7, 1958, at the New York Eye and Ear Infirmary, 218 Second Ave., New York 3. Registration is limited to 15, at a fee of $100 each. The slides and specimens used will be drawn from a large collection of material. For further information, address the Registrar, Institute of Ophthalmology of the Americas at the above named hospital.

The fifth June workshop on **Developing Concepts in Community Rehabilitation Services** is scheduled for June 2-20, 1958, at the Institute for the Crippled and Disabled, 400 First Ave., New York 10, in affiliation with Columbia University. The intensive course for physiatrists, psychiatrists, occupational and physical therapists, social workers, and others active in rehabilitative health services will consist of lectures, seminars, clinical demonstrations, and problem clinics at a fee of $115, if graduate credit is desired, or $100 otherwise. A limited number of subsistence stipends are available to United States citizens who could not otherwise attend. Further information may be obtained from Director of Professional Education, Institute for the Crippled and Disabled.

An intensive four-day, full-time course, **Fundamentals of Psychiatry and Psychosomatic Medicine—Principles of Diagnosis and Treatment,** will be presented by the Psychiatric Institute of the University of Maryland School of Medicine and the National Institute of Mental Health, U. S. Public Health Service, June 9-12, 1958, at the Psychiatric Institute, Baltimore. The first two days will consist of lectures and demonstrations with patients, registrant participation, and panel discussion. The final two days, with enrollment limited to 20 physicians, will provide opportunity for supervised patient interviews by registrants. Further information may be obtained from Postgraduate Committee, University of Maryland School of Medicine, 522 W. Lombard St., Baltimore 1.

The Massachusetts Institute of Technology will offer an intensive course for research workers in biology and medicine July 8-18, 1958, **Modern Research Methods in Biology and Medicine.** By means of lectures, demonstrations, general discussions, guided tours, and visits to research laboratories, an intensive introduction will be provided to physical research methods and instruments applicable to biological and medical research, at a tuition of $250. Further information may be obtained from Dr. Kurt S. Lion, Biophysics Department, Massachusetts Institute of Technology, Cambridge 39, Mass.

MEDICINE AND THE LAW

MEDICAL DEFENSE IN UNITED KINGDOM

It is now well established that since the advent of a comprehensive National Health Service on July 6th, 1948, medical litigation in the United Kingdom has increased threefold. The payments made by hospital authorities in compensation of all kinds in England and Wales during the material years are as follows: 1948-1949 £7,500, 1950 £23,000, 1951 £38,000, 1952 £106,000, 1953 £153,000, and 1954 £159,000. These figures do not take into consideration the very large sums paid by defense organizations in unsuccessful suits at law or in settlements out of court.

Persons who have examined the professional liability problem have attributed the increase in litigation to several factors. First of all, they say that the advent of the National Health Service has contributed materially to the increase. Under that service all citizens, with certain specific exceptions, are obliged to pay, directly and indirectly, for the provision of a national general practitioner and hospital service, including consultants and specialists of all kinds and special forms of therapy. Prior to 1948, the benefits of the National Health Service then in existence were of a limited order and exclusive of hospital services; the beneficiaries also were limited in number and financial status.

Compulsory Contribution

Since a health service now exists which is available to all, it is not surprising that the pressure on doctors, nurses, and others causes conflict and disagreement between physicians and patients from time to time. Allegations of wrong treatment, of wrong diagnoses, and of other forms of professional negligence are associated with adverse criticism and unsatisfactory clinical results.

Disappearance of Voluntaryism

The hospitals are no longer voluntary institutions maintained by voluntary workers with grants from private local and national organizations. The medical and surgical staff no longer provide gratuitous services. The spirit of voluntaryism has disappeared. The staff are now fully paid for the services they render. As a consequence, aggrieved or disgruntled persons regard these state hospitals as possessing a bottomless purse into which they may dip for the payment of compensation when they have a real or imaginary cause for complaint.

Legal Aid and Advice Act, 1949

Another factor that has doubtless increased medical litigation is the passing of the Legal Aid and Advice Act, 1949. Under that act a scheme has been instituted in the United Kingdom whereby people of a certain economic status are enabled to litigate at little or no cost to themselves. The prospective litigant must merely satisfy a local committee that there is a prima facie case warranting investigation by a court. For this purpose, in a professional liability case, a report is obtained from one medical practitioner on the work of another. On this evidence the committee reaches a decision as to whether or not to issue a legal aid certificate for the benefit of the prospective plaintiff. Nearly 80% of all cases of medical litigation held in the United Kingdom involve plaintiffs holding a legal aid certificate.

It should not be thought, however, that there is an absence of justification for the legal aid scheme. Indeed, figures published by the law society are demonstrative of the fact that the scheme serves a social purpose that must have been crying for solution for some time past. Taking the figures published in *The Times* of Feb. 24th, 1955, the law society announced that in the previous five years applications for legal aid in all cases—not only medical cases—exceeded 200,000. The great majority of these litigants won their cases and recovered over £6,000,000 in damages. For the past three years, the report went on, the percentage of success attained by legally aided litigants was in the neighborhood of 89% of the cases brought in the Queen's Bench division, and a little lower in the Chancery division. In the court of appeal the figure of successful appeals was 53%.

It is not accurate, as is usually done, to describe the scheme as "free legal aid," as in 63% of the cases some contribution is made by the litigant. Indeed, in the first four years of its operation, litigants contributed over £2,000,000, and over £1,000,000 was contributed by unsuccessful opponents. The state contributed nearly £2,500,000.

A further factor contributing to the increase of medical litigation derives from a decision of a court of appeal whereby hospital authorities are made responsible for the acts or omissions of the members of their staffs in the pursuit of their professional and technical duties for which they are engaged. Where many litigants would hesitate to sue their own family doctor or a popular surgeon or physician who has helped them towards recovery, they have no hesitation in suing a regional hospital board or a hospital management committee or a board of governors. These institutions represent a collection of individuals with high resounding names and considerable powers, as well as the backing of the Ministry of Health, and behind that the treasury of the state. To sue such a body is not

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likely to cause the qualms of conscience that would occur if the proceedings were directed against an individual surgeon or physician. They have neither a body to kick nor a soul to be damned.

Negligence

The standard of care required of a medical practitioner was fully expressed by a Lord Chief Justice nearly 100 years ago in the case of *Rich* v. *Pierpoint* and, 26 years later, in the case of *Lanphier* v. *Phipos*. It reads as follows.

> Every person who enters into a learned profession undertakes to bring to the exercise of it a reasonable degree of care and skill. He does not undertake, if he is an attorney, that at all events you will gain your case, nor does a surgeon undertake that he will perform a cure, nor does he undertake to provide the highest possible degree of skill. There may be persons who have higher education and greater advantages than he has, but he undertakes to bring a fair, reasonable, and competent degree of skill. Practitioners may be guilty of mistakes and, indeed, often are. They may be guilty of effecting a wrong diagnosis, of tendering wrong advice, and indeed there may ensue some unfortunate catastrophe. The test ultimately shall not be whether a mistake was committed, but whether the practitioner exercised all that degree of care and skill which a reasonable man would expect him to exercise, and which he was competent to exercise. If he has done all that he should and could do and left undone nothing that he should and could do, it is highly improbable that he would be found guilty by any Judge or Jury.

Since that time a series of cases have been heard and decided in the English and Scottish courts affecting general practitioners and hospital medical officers. For those who are interested, there is a clear progress of evolution from the attitude adopted toward hospital medical officers in the early part of this century and that adopted toward them and their employers at the present time. To discuss these several cases in their sequence would entail considerable space and certainly more than is available or warrantable in this article. Reference to the legal and professional press could be made by those who desire to pursue this matter in further detail. Suffice it to say, however, that whereas in the early part of the century the governing body of a hospital was not thought to be liable for the acts of omissions of the hospital staff, save when they were engaged in a ministerial capacity, it has now been held by a deciding case of commanding importance that the employing authority does carry that responsibility.

The machinery also exists whereby the employing authority can serve a notice of contribution on the medical employee, or bring him in as a third party and thus point to him, or recover from him, as the negligent person, the damages awarded by way of compensation for the wrong suffered by the patient.

It follows from this that it is no less important today than it was 80 years ago for there to be in existence an appropriate medical organization to advise, protect, and indemnify a doctor when he is confronted with medical litigation.

Medical Defense Union

The telegraphic address of The Medical Defense Union in London is "Damocles." This word was expressly chosen to symbolize the medicolegal risk confronting every medical practitioner in his professional life. It is difficult for the modern practitioner to realize the state of affairs that existed before the Medical Defense Union was constituted to mitigate the hardships of the law as it then pertained to medical practice. In those days if a practitioner was prosecuted for negligence or malpractice, he had to arrange for his own defense and bear the cost of it himself; and if he were the victim of slander or libel he had to sue his persecutor at his own expense or suffer in silence. His case, however good, was apt to be prejudiced by lack of expert advice and, even if were successful in the courts, he might find himself in severe financial straits owing to the costs of the action. It was not uncommon for appeals to be made in the medical press for funds to help him in his difficulties, and it says much for the generosity of the medical profession that those were rarely made in vain.

During the 1880's it occurred to some members of the profession that attacks on medical practitioners were increasing in number and that many prosecutions, or threats of them, were animated by malice or based on frivolous pretexts. Two cases especially roused the indignation of the profession. In one of these two Dulwich practitioners, Dr. Bower and Dr. Keates, were subjected to a series of prosecutions and lawsuits which the *British Medical Journal* characterized as "very little on the hither side of vexatious litigation." The practitioners had performed a tracheotomy on a child found, after a provisional diagnosis of croup, to be suffering from diphtheria, and they instructed the father to suck the tube to remove any obstruction. The child died and the father contracted diphtheria. Subsequently the father instituted a charge of manslaughter against the two doctors, and after a five-day hearing in November, 1883, the magistrate dismissed the case without calling upon the counsel for the defense, remarking that "if he allowed the enquiry to proceed further, he would not only be sanctioning a prosecution, but a persecution as well, and that it would be next to wickedness on his part to commit, as he could not see one tittle of evidence to support the allegations set forth in the indictment." Undeterred by this denunciation the father then brought an action for negligence on the ground that the practitioners should have diagnosed diphtheria earlier and should have given a warning of danger of infection incurred by sucking the tracheotomy tube. There were two trials because the first jury could not agree. In the second trial the jury, after hearing the plaintiff's statement, thought it unnecessary to hear the defendants' counsel. Although the doctors were

successful they incurred costs amounting to over £1,000 and had to endure the anxiety of long, drawn-out proceedings. Early in the case an appeal for funds to defray the expenses was launched by Sir William Jenner, and to this the profession responded with promptitude and liberality.

The second case was a more painful one, for the accused practitioner actually served eight months of a sentence of two years' imprisonment with hard labor before he was released with a free pardon. In November, 1884, Dr. David Bradley of Chesterfield was convicted of an attempt to commit a criminal assault on a woman patient. The profession clamored for his release and a memorial was submitted to the Home Secretary expressing the view that the evidence failed to support the charge and that due weight had not been given to the fact that the patient had been subject from childhood to epileptic fits and was liable to have delusions. It was generally believed that a conviction would never have been secured if adequate and available expert evidence had been submitted on Dr. Bradley's behalf. Eventually, the Home Secretary, on being satisfied that there was sufficient doubt of Dr. Bradley's guilt to warrant the granting of a free pardon, directed the prisoner's release. Again Sir William Jenner initiated a fund to help an unfortunate colleague to reestablish himself in practice after his tragic ordeal.

Such cases as these convinced medical practitioners of the very real risk they ran in their daily routine, and it was felt that the raising of funds after the event was not the ideal solution of the problem. The Medical Defense Union was thereupon born, albeit of lay parentage, on Oct. 23, 1885. It was registered as a company limited by guarantee and its Memorandum listed the following objects as being those for which it was established:

(i) To support and protect the character and interests of medical practitioners practising in the United Kingdom.

(ii) To promote honourable practice and to suppress or prosecute unauthorized practitioners.

(iii) To advise and defend or assist in defending members of The Union in cases where proceedings involving questions of professional principle or otherwise are brought against them.

(iv) To consider, originate, promote and support (so far as is legal) legislative measures likely to benefit the medical profession and to oppose all measures calculated to injure it: and for the purposes aforesaid to petition Parliament and take such other steps and proceedings as may be deemed expedient.

The promoters envisaged a membership of many thousands, an annual subscription of 10 shillings, a large reserve fund derived from members' guarantees and, possibly, an entrance fee.

The persons responsible for initiating the organization were all laymen, and the response given by the profession to the new body was mixed; some welcomed it, and others had doubts about its intentions. Some thought that a defense body should be organized by and within the profession, especially by the British Medical Association, but this was held to be impossible by the constitution of the association. One or two efforts made to bring medical defense within the scope of the benefits of members of the association have hopelessly failed.

Various branches were formed in different parts of the country, and in some of these local solicitors were appointed to act when instructed on behalf of the union. It was intended that these local councils should examine local applications for assistance. They were urged to ascertain the antecedents, position, and character of the accuser as well as the evidence he held to substantiate his claim. It was incumbent upon the local representative to formulate his recommendation for submission to the General Secretary, who would initiate the necessary machinery for the protection of the practitioner involved.

It would take a long time to describe the stormy experiences that the union underwent in its early years. Perhaps this is not the occasion to do so. Suffice it to say that with the passage of time the numbers joining the union grew year by year. A central council was appointed with a president and vice-president. The local branches were eventually disbanded. The whole of the administration and power was concentrated in the central body. Offices were taken in London, a secretary was appointed, and the infant grew from adolescence to adult status among the many professional bodies operating in London; long before it had reached its majority it had become firmly established. The fame of the union crossed the seas and the profession in New Zealand established a defense organization modeled on it. Requests for advice on establishment of other bodies were received from Canada and the United States of America. It handled libel and slander cases and malpractice actions, prosecuted unqualified persons, and gave much individual advice to members on miscellaneous problems raised in correspondence.

Its membership is now in excess of 45,000 medical practitioners and a small number of dentists. Its annual subscription for home members for unlimited indemnity does not exceed £3 per annum; an additional subscription is paid by members resident and practicing overseas, varying with the amount of financial protection desired. The union does not accept members in the United States and in some other parts of the world.

It is the firm belief and conviction of all who have studied its progress and its work that the union is indispensable to the medical profession of the United Kingdom. It is a dignified and highly accepted professional institution that has earned the authority to offer its hand to the doctor as Everyman and say

> I will go with thee, and be thy guide,
> In thy most need to go by thy side.

MEDICAL NEWS

ARIZONA

State Meeting in Chandler.—The 67th annual meeting of the Arizona Medical Association will be held at the San Marcos Hotel, Chandler, April 30-May 2. The faculty for the meeting includes Drs. Cyril M. MacBryde and Louis T. Byars, St. Louis; James F. Nolan and George C. Griffith, Los Angeles; William R. Arrowsmith, New Orleans; William C. Deamer and Robert S. Pollack, San Francisco; Juan A. Del Regato, Colorado Springs, Colo.; and Charles W. Elkins, Tucson. About 18 papers are scheduled, with question and answer periods to follow the presentations. Motion picture sessions and specialty luncheons are planned for the afternoons of May 1 and 2. The dinner dance is scheduled for May 2, 7:45 p. m. Entertainment includes a golf tournament May 3. For information write Dr. Leslie B. Smith, 826 Security Bldg., Phoenix, Ariz.

FLORIDA

Occupational Medicine Conference.—The second Conference on Occupational Medicine, sponsored jointly by the Medical Schools of the Universities of Havana, Cuba, and Miami, Florida, will be held in Miami, Aug. 18-22. William B. Deichmann, Ph.D., professor of pharmacology at the University of Miami School of Medicine, Coral Gables, is chairman of the conference, and Dr. M. Eugene Flipse, associate professor of medicine, is chairman of the program committee. Dr. Francisco Lancis y Sanchez, president of the Cuban Industrial Medical Society, and Dr. Rafael Penalver Ballina, of the University of Havana School of Medicine, are members of the planning committee. Proceedings of the conference will be in Spanish. Inquiries on attendance or papers may be referred to Dr. Deichmann.

ILLINOIS

State Anesthesiologists Meet in Chicago.—The annual meeting of the Illinois Society of Anesthesiologists will be held May 18 at the Hotel Sherman, Chicago. The morning will be devoted to a symposium, "Economic Problems in the Practice of Anesthesia," moderated by Dr. William O. McQuiston, Peoria, and including the following panelists: Drs. Wayne B. Slaughter, clinical professor of surgery, University of Wisconsin Medical School, Madison; Lindon Seed, Robert A. Beebe, and George Z. Wickster, Chicago; and Lawrence D. Ruttle, Joliet. In the afternoon, Drs. Morton D. Leigh, of Los Angeles, and Kenneth K. Keown, of Columbia, Mo., will present "Monitoring Devices in Pediatric Anesthesia" and "The Role of the Anesthesiologist in the Treatment of Coronary Disease," respectively. A business meeting and election of officers will follow the scientific program. For information write Dr. Huberta M. Livingstone, 5805 Dorchester, Chicago 37.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education, and public health. Programs should be received at least three weeks before the date of meeting.

Chicago

Dr. Jenkinson Honored.—Former residents of Dr. Edward L. Jenkinson have honored their chief, who retired last September as professor of radiology and chairman of the department at Northwestern University School of Medicine, with establishment of "The Edward L. Jenkinson Fellowship in Radiology" at the Medical School. Dr. Jenkinson will retire in May after 41 years as director of the department of radiology, St. Luke's Hospital.

New Hospital at County Jail.—The new 50-bed hospital at Cook County jail was opened Feb. 3. The hospital was built with funds from regular maintenance accounts and with labor volunteered by maintenance men. A basement area of the jail, formerly used as a dormitory, was converted to create a 24-bed isolation ward for tuberculous patients, a 22-bed general ward, and a four-bed contagious disease ward. It has an operating room, x-ray room, receiving ward, oxygen equipment, and dental laboratory. Over 90% of inmate medical needs will be met by the new hospital facilities. The staff will include a full-time dentist, four part-time physicians, a part-time psychiatrist, a surgeon on 24-hour call, two full-time orderlies, six nurses, a lab technician, and three externs. Dr. Karl A. Meyer, medical superintendent, Cook County Hospitals, gave the dedication address.

MISSOURI

Premiere of Symphonic Overture.—The world's premiere of "Heloise et Abelard," a symphonic overture-fantasy written by Dr. Vincent T. Williams, of Kansas City, was performed recently over station WDAF, Kansas City, by Hans Schwieger and the Kansas City Philharmonic Orchestra.

Personal.—Dr. Albert B. Eisenstein has been appointed full-time director of the Division of Medicine at the Jewish Hospital of Saint Louis,

effective July 1, succeeding Dr. Sol Sherry, who is assuming a position as professor of medicine at the Washington University School of Medicine.——Dr. Ferdinand C. Helwig was honored recently when a dinner was held "marking his thirty years of association with St. Luke's Hospital."

Society News.—New officers of the St. Louis Academy of General Practice for 1958 are Drs. Walter C. Gray, president; Eugene W. Hall, vice-president; Charles A. Molden, president-elect; Vernon E. Michael, treasurer; Eugene V. Henschel, secretary.——The new officers of the Greater St. Louis Radiological Society are as follows: president, Dr. Sam J. Merenda; vice-president, Dr. Thomas F. Maher; and secretary-treasurer, Dr. Charles J. Cherre.

NEW YORK

Fellowship for Muscle Disease Research.—Award of the first McEachern Postdoctoral Fellowship, a $6,500 stipend for advanced training and research in the area of muscle disease, has been given to Jerald L. Connelly, Ph.D., currently a graduate fellow in the department of biochemistry, University of Rochester School of Medicine and Dentistry. Dr. Connelly's research will be conducted at the Institute for Enzyme Research, University of Wisconsin, Madison. The university has been granted $1,000 for laboratory maintenance in connection with Dr. Connelly's work, which will be under the supervision of Henry A. Lardy, Ph.D., professor of biochemistry. The fellowship is one of two established by the Muscular Dystrophy Associations of America, Inc., to provide opportunities for scientists who wish to follow careers in research or academic medicine. It was named in honor of the late Dr. Donald McEachern, associate professor of neurology at McGill University, Montreal, Canada, and a member of the associations' Medical Advisory Board. The other fellowship, named for the late Dr. Joseph L. Lilienthal, was awarded for the first time in November of last year.

New York City

Continue Study of Heredity in Polio.—Columbia University will continue studies of the mechanism that controls heredity in polio viruses under a grant of $70,226 from the National Foundation for Infantile Paralysis. The work, under the direction of Dr. Hattie E. Alexander, associate professor of Pediatrics, is being carried out at Babies Hospital, where Dr. Alexander is attending pediatrician. Her co-workers are Isabel Morgan Mountain, Ph.D., research associate, department of microbiology, who has been assigned to Pediatrics; Dr. Katherine Sprunt, research associate in pediatrics, and Dr. Gebhard Koch, of Germany, who has been invited to participate in the group.

Personal.—Dr. George E. Wakerlin has been appointed medical director of the American Heart Association. Dr. Wakerlin comes to the association from the University of Illinois College of Medicine, Chicago, where he has served as head, department of physiology, since 1937.——On March 1 Dr. Jesse D. Stark was elected a member of the Institute of Humanities of the Republic of France and awarded the Medal for Merite Scientifique.——The Western Electric Company announces the appointment of Dr. Edward J. Schowalter as medical director. In this newly created post, Dr. Schowalter will be responsible for over-all development and coordination of medical and health programs throughout the company. He assumed his new duties March 1.

OKLAHOMA

State Medical Meeting in Oklahoma City.—The Oklahoma State Medical Society will hold its 52nd annual meeting May 5-7 at the Municipal Auditorium, Oklahoma City. Eleven out-of-state speakers will present papers: Drs. Edwin L. Prien, Boston; Jacob A. Bargen and Charles W. Mayo, Rochester, Minn.; John E. Hobbs, St. Louis; Edgar J. Poth, Galveston, Texas; Charles H. Brown, Cleveland; Alvin J. Ingram, Memphis, Tenn.; Robert D. Moreton, Fort Worth, Texas; Kenneth C. Johnston, Chicago; Louis A. Soloff, Philadelphia; and John H. Githens, Denver. Thirty scientific and organizational exhibits will be located in the Zebra Room of the Auditorium. A physicians hobby show, technical exhibits, and round-table luncheons are planned. The president's inaugural dinner-dance will be held May 6, at 7 p. m. A golf tournament is scheduled for May 7. For information write the Oklahoma State Medical Association, Box 9696, Oklahoma City, Okla.

OREGON

Quartet Wins Competition.—For the third time the Forcep Four, made up of University of Oregon Medical School students (and one graduate) won the annual Original All Northwest Barber Shop Ballad Contest, held Feb. 21-March 1. More than 3,000 persons turned out for the competition. The group includes Al Witt, senior, first tenor; Doug Day, senior, baritone; Ben Shepherd sophomore, second tenor; and Dr. W. Robert Day, second-year pediatrics resident, bass. The group, using live animals for "props," delighted the audience with "Jungle Town." The winners took home $300, individual trophies, and a large perpetual trophy.

PENNSYLVANIA

Summer Workshops in Health Education.—Health education workshops aimed at "a more effective community-school health education program" have been scheduled for six Pennsylvania colleges and universities this summer under the auspices of the

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Inter-Agency Planning Committee, which includes the Medical Society of the State of Pennsylvania, the Pennsylvania Department of Health, Pennsylvania Department of Public Instruction, Pennsylvania Department of Welfare, and Pennsylvania Tuberculosis and Health Society. Enrollment is open to teachers, nurses, parents, school administrators, dental hygienists, guidance personnel, nutritionists, agency administrators, and public health personnel. A limited number of scholarships are available. The location of the workshops and the dates include Pennsylvania State University and Slippery Rock State Teachers College, June 9-27; Lehigh University, June 16-July 3; University of Pittsburgh and West Chester State Teachers College, June 23-July 11; and Temple University, June 30-July 18.

RHODE ISLAND

Annual State Medical Meeting.—The annual meeting of the Rhode Island Medical Society will be held May 13-14 in Providence. Dr. George W. Waterman, of Providence, president of the society, will present "Problems Facing the Medical Profession" the afternoon of May 14. Topics to be presented by out-of-state speakers include:

Clinical and Experimental Homotransplantation of Skin, Kidney, and Bone Marrow, Dr. Joseph E. Murray, Boston.
The Prevention of Somatic and Genetic Radiation Injury, Dr. Shields Warren, Boston.
Surgical Problems of the External Biliary Tract, Dr. Leland S. McKittrick, Boston.
Hiatus Hernia and Esophagitis, Dr. Richard H. Marshak, New York City.
When Should Heart Disease be Compensable, Dr. William E. R. Greer, Boston.
Neurosurgical Methods of Relieving Severe Chronic Pain in the Thorax and Abdomen, Dr. James C. White, Boston.

Other speakers are Drs. Americo Savastano and Henry C. McDuff, of Providence. For information write the Rhode Island Medical Society, 106 Francis St., Providence 3, R. I.

TENNESSEE

Dr. Grigorieff Goes to Vienna.—Wladimir W. Grigorieff, Ph.D., chairman, University Relations Division, Oak Ridge Tennessee Institute of Nuclear Studies, will take a one-year leave of absence to become chief of the Exchange Branch of the International Atomic Energy Agency Division of Exchange and Training of Scientists and Experts. He will leave Oak Ridge May 15 to assume his new duties June 1 in Vienna. The IAEA, formally inaugurated in Vienna in October, 1957, is an outgrowth of a proposal made by President Eisenhower four years previously. The agency is an international organization independent of any national government. Its purposes are to accelerate and enlarge the contribution of atomic energy to peace, health, and prosperity throughout the world, and to ensure that the assistance it provides is not used to further any military purposes. The IAEA staff is made up of representatives of many nations. Dr. Grigorieff has been national executive secretary of the American Nuclear Society since September, 1955.

UTAH

Ogden Surgical Meeting.—The 13th annual meeting of the Ogden Surgical Society will be held May 21-23 at the Orpheum Theatre, Ogden. Guest speakers include Drs. Harry E. Bacon, Philadelphia; Benjamin M. Black, Rochester, Minn.; Alexander Brunschwig and Gerald H. Pratt, New York City; William Dameshek and Claude E. Welch, Boston; Richard K. Gilchrist, Chicago; Roy G. Holly, Omaha; James F. Nolan, Los Angeles; Alton Ochsner, New Orleans; Col. Joseph R. Shaeffer, M. C., U. S. Army, Washington, D. C.; Col. Arthur B. Tarrow, M. C., U. S. A. F., Lackland Air Force Base, Texas; Owen H. Wangensteen, Minneapolis; and James V. Warren, Durham, N. C. A program of entertainment and a ladies' program are arranged. For information write to Dr. Leath D. Nelson, Washington Terrace, Ogden, Utah.

WISCONSIN

Annual State Meeting in Milwaukee.—The 117th annual meeting of the State Medical Society of Wisconsin will be held May 6-8 at the Milwaukee Auditorium and Hotel Schroeder. The Lucy Droessel Memorial Lecture of the Wisconsin Heart association will be presented by Dr. John W. Wilce, of Columbus, Ohio, on "Cardiac Effects of Athletic Work Stress." Papers presented by out-of-state speakers at the general sessions include:

The Organic Explanation of Some Behavior Disturbances, Dr. John C. MacQueen, Iowa City.
Oral Preparation for the Treatment of Diabetes, Dr. Howard F. Root, Boston.
Cancer Detection in the Office of the Generalist, Dr. John S. De Tar, Milan, Mich.
Perinatal Adjustments, Dr. Virginia Apgar, New York City.
The Life and Work of William Beaumont, Dr. Burrill B. Crohn, New York City.

Panel discussions, round-table luncheons, and scientific and technical exhibits are planned. Special functions include a golf tournament, the annual dinner (May 7), and the president's reception. For information write the State Medical Society of Wisconsin, Box 1109, Madison 1, Wis.

Society News.—Recently elected officers of the Wisconsin Radiological Society are: president, Dr. William W. Moir, Sheboygan; president-elect, Dr. Samuel R. Beatty, Neenah; secretary-treasurer, Dr. Farrell F. Golden, 5221 Tonyawatha Trail, Madison.—The executive offices of the Medical Society of Milwaukee County are now located at 756 N. Milwaukee St., Milwaukee 2, Wis.—The Wisconsin Society of Anesthesiologists has installed the

following officers: president, Dr. Loren F. Thurwachter Jr., Milwaukee; president-elect, Dr. James W. Bookhamer, Milwaukee; and secretary-treasurer, Dr. Betty J. Bamforth, Madison.——The Brown-Kewaunee–Dorr County Medical Society was reorganized in 1957 into two separate societies: Brown County Medical Society, with Dr. Frank Urban, secretary, 302 Northern Building, Green Bay; and Dorr–Kewaunee County Medical Society, with Dr. Jack F. March, secretary, 413 4th St., Algoma.

Personal.—Dr. W. Theodore Bruns, has been named assistant director of the new Respirator Center under organization at the University of Wisconsin Hospitals. Last November the university regents approved appointment of a director for the Respirator Center, Dr. Arthur A. Siebens, who will join the staff April 1.——Dr. William B. Parsons Jr. has been appointed director of research of the Jackson Foundation, an institution organized in 1947 for medical research and education and housed in the Jackson Clinic Building, Madison. The foundation has recently received grants from the National Heart Institute, the American Heart Association, the Wisconsin Heart Association, and the Henry K. Wampole Laboratories, made to Dr. Parsons for his studies on the relationship between blood cholesterol and arteriosclerosis.——Dr. Gerald C. Mueller, department of oncology, University of Wisconsin Medical School, Madison, has left for several months of research in European laboratories under a George I. Haight Traveling Research Fellowship. Dr. Mueller's plans include virus research at the Max Planck Institute für Virusforschung, Tübingen, Germany, and visits to laboratories in England and Scandinavia. The University Research Committee selects the Haight fellows.

HAWAII

Annual Meeting in Honolulu.—The Hawaii Medical Association will hold its annual meeting April 30-May 4, in Honolulu. The program includes a discussion, "Some Recent Developments in Surgery," moderated by Dr. Clarence M. Burgess, and three panel discussions: Recent Advances in Internal Medicine, Recent Advances in Pediatrics, and Radioactive Isotopes. A four-man demonstration of recent developments in psychotherapy that can be used in practice will be given the morning of May 2, with an illustrated discussion on medical photography to be held that evening. Scientific and technical exhibits are planned, and entertainment includes a golf tournament (May 4) and a dinner dance (May 3). For information write the Hawaii Medical Association, 510 S. Beretania St., Honolulu 13, Hawaii. Members of the American College of Obstetricians and Gynecologists, whose meeting is being held in Los Angeles preceding the Hawaii meeting, are invited.

GENERAL

New Science Information Division.—Rolland J. Main, Ph.D., has been named director of the new Scientific Information Division of Eaton Laboratories, Norwich, N. Y. This new division will be part of the Scientific Department under the over-all supervision of William B. Stillman, Ph.D., research administrative coordinator. Dr. Main's division will collect, catalogue, and correlate all published and unpublished information on the company's products. It will also translate all pertinent scientific literature published throughout the world. The new division will service Eaton's research and medical departments and physicians. Its staff includes medical editors, translators, literature scanners, and medical and research librarians. Dr. Main, who was professor of physiology and lecturer in pharmacology, Medical College of Virginia, Richmond, for 15 years, will continue to supervise Eaton's medical information services.

Society News.—The officers of the **Central Surgical Association** are: president,Dr. William A. Altemeier, Cincinnati; president-elect, Dr. Charles D. Branch, Peoria, Ill.; secretary, Dr. Angus D. McLachlin, London, Ontario; treasurer, Dr. Chester B. McVay, Yankton, S. D.; and recorder, Dr. Robert T. Tidrick, Iowa City.——At its 35th annual meeting the **American College of Radiology** elected the following new officers: president, Dr. Vincent W. Archer, Charlottesville, Va.; vice-president, Dr. Ivan M. Woolley, Portland, Ore.; and elected to four-year terms as chancellors on the board of chancellors, Drs. Harold O. Peterson, St. Paul, Minn., and John A. Evans, New York City.——Officers of the **Society of American Bacteriologists** for 1958 are: president, Dr. Harry Eagle, Bethesda, Md.; vice-president, Philip R. Edwards, Ph.D., Chamblee, Ga.; secretary, E. M. Foster, Ph.D., Madison, Wis.; and treasurer, John Hays Bailey, Dr.P.H., Rensselaer, N. Y.

Medical Science Scholars.—The John and Mary R. Markle Foundation announces the appointment of 25 Scholars in Medical Science, all faculty members of medical schools in the United States and Canada. The sum of $750,000 has been appropriated toward their support to the schools where they will teach and do research. For each scholar appointed, the Foundation has allocated $30,000 to the school at the rate of $6,000 a year for five years. The purpose of the program is "to strengthen medical education by offering academic security and financial help to teachers and investigators in medical schools early in their careers." In the 11 years the plan has been in operation, 231 doctors in 74 medical schools in the U. S. and Canada have received help from appropriations totalling $6,800,000. The 25 scholars were selected from 57 candidates nominated by medical school deans. The foundation was established in 1927 by the late John Markle,

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Pennsylvania coal operator, "to promote the advancement and diffusion of knowledge . . . and the general good of mankind."

National Meeting of Clinical Research Federation.—The American Federation for Clinical Research will hold its national meeting May 4 at the Steel Pier Theatre, Atlantic City, N. J. Foreign participants include Drs. Julien I. E. Hoffman and George G. Rowe, of London, England, who will present "Some Factors Affecting Indicator Dilution Curves in the Presence and Absence of Valvular Incompetence," and Oliver M. Wrong and Howard E. F. Davies, of Manchester, England, presenting "The Excretion of Acid in Renal Disease." Section meetings under joint sponsorship of the federation and the American Society for Clinical Investigation will be held in the afternoon and evening. Presiding will be Drs. James V. Warren, Laurence H. Kyle, Arnold S. Relman, Lawrence E. Young, Samuel P. Martin, III, Albert I. Mendeloff, and Robert R. Wagner. For information write Dr. William W. Stead, Veterans Administration Hospital, 54th Street and 48th Avenue South, Minneapolis 17, Minn.

Neurologists Annual Meeting in Philadelphia.—The 10th annual meeting of the American Academy of Neurology will be held April 21-26 at the Bellevue-Stratford Hotel, Philadelphia. The Robert Wartenberg Memorial Lecture, "The Pathological Basis of Chorea and Choreoathetosis," will be given by Dr. Joseph G. Greenfield, London, England. Over 60 papers are scheduled for presentation at the general sessions, including "The Carcinomatous Neuromyopathies," by Sir Russell Brain, London, England. About 54 papers will be read by title. Symposiums on Pharmacological Stimulation of the Central Nervous System, Neurochemistry of Amino Acids and of Nucleotides, and Higher Brain Functions are planned. A separate meeting of the Section of Neuroanatomical Sciences will be held April 24. Movies and scientific and technical exhibits are planned. The annual banquet will be held April 25. For information write the American Academy of Neurology, 3501 E. 54th St., Minneapolis 17, Minn.

Meeting of Bacteriologists in Chicago.—The 58th general meeting of the Society of American Bacteriologists will be held under the auspices of the Illinois Branch April 27-May 1 at the Morrison Hotel, Chicago. Over 400 papers are scheduled for presentation at the five major division meetings, and 38 will be read by title. Five symposiums are planned: Animal Viruses and Tissue Culture, Problems of Cell Permeability, Effects of Broad Spectrum Antibiotics on Cell Metabolism, Bacterial Cell Walls, and Staphylococcal Infections. A special lecture, "The Key to Lockjaw," will be given April 27 by W. E. van Heyningen, of the University of Oxford, England. The presidential address will be given the afternoon of April 28 by Dr. Harry Eagle, of the National Institutes of Health. Round-table discussions, scientific and technical exhibits, six motion pictures, and tours are planned. The president's reception and dinner will be held April 29. For information write the Society of American Bacteriologists, Dr. Edwin M. Foster, Secretary, University of Wisconsin, Madison, Wis.

Fellowships for Mental Health Training of Clergy.—The Academy of Religion and Mental Health has announced a grant of $10,000 from the Smith, Kline and French Foundation to provide fellowships for theological students and clergymen of all faiths who want to become chaplains in mental hospitals. A grant of a similar amount for the same purpose was given in 1957. The program will be known as the Smith, Kline and French Foundation Chaplaincy Fellowships. The foundation is the independent philanthropic arm of the Laboratories. The Academy of Religion and Mental Health in a nonsectarian, nonprofit organization founded in 1954 "to develop further relationships between religious groups and the mental health professions." Its membership includes 1,200 members of the American Psychiatric Association, 500 members of the American Psychological Association, 800 clergymen, and more than 400 psychiatric social workers, cultural authropologists, educators, and laymen. It serves as a consultant to hospitals, medical schools, and mental health organizations in this country and abroad.

Medical Lecture Tour to Asia.—The Asia-Pacific Academy of Ophthalmology is sponsoring a good will tour to countries of the Orient following the International Congress of Ophthalmology in Brussels in September. The purpose of this tour, which is to last about one month, is to hold joint meetings with ophthalmologists in Pakistan, India, Thailand, the Philippines, and Hong Kong. The Asia-Pacific Academy of Ophthalmology was organized in 1957. Its principal purposes are "to extend ophthalmologic knowledge and to advance the arts and sciences of ophthalmology and related sciences in Asia and in countries bordering the Pacific Ocean; . . . to stimulate research in tropical and systemic eye diseases, that are particularly prevalent in Asia and in countries bordering the Pacific Ocean; to cultivate social and fraternal relationship of physicians residing in Asia; . . . to offer postgraduate instruction in ophthalmology through the medium of lectures, round-table discussions, seminars, clinics, films and other means." Physicians other than ophthalmologists and their families are invited. Those desiring to participate in the postgraduate lectures and seminars on medical subjects pertinent to

ophthalmology should contact Dr. William John Holmes, Liaison Secretary, Suite 280, Alexander Young Building, Honolulu 15, Hawaii.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States, its territories and possessions in the weeks ended as indicated:

Area	March 29, 1958 Paralytic Type	March 29, 1958 Total Cases	March 30, 1957 Total
New England States			
Maine	..	..	..
New Hampshire	..	..	..
Vermont	..	..	..
Massachusetts	..	..	..
Rhode Island	..	..	..
Connecticut	1	1	..
Middle Atlantic States			
New York	..	..	1
New Jersey	..	..	..
Pennsylvania	..	..	..
East North Central States			
Ohio	..	..	..
Indiana	..	1	..
Illinois	..	..	..
Michigan	..	..	..
Wisconsin	..	..	1
West North Central States			
Minnesota	..	..	..
Iowa	..	..	..
Missouri	..	..	..
North Dakota	..	..	..
South Dakota	..	..	..
Nebraska	..	..	3
Kansas	..	..	1
South Atlantic States			
Delaware	..	..	..
Maryland	..	..	..
District of Columbia	..	..	..
Virginia	..	..	3
West Virginia	..	..	..
North Carolina	..	..	..
South Carolina	..	..	..
Georgia	..	..	..
Florida	1	1	1
East South Central States			
Kentucky	..	..	..
Tennessee	1	1	..
Alabama	..	..	2
Mississippi	..	..	1
West South Central States			
Arkansas	..	..	..
Louisiana	..	..	2
Oklahoma	1	2	..
Texas	..	1	4
Mountain States			
Montana	..	..	..
Idaho	..	..	..
Wyoming	..	1	..
Colorado	2	3	1
New Mexico	..	..	..
Arizona	..	..	..
Utah	..	..	..
Nevada	..	..	..
Pacific States			
Washington	..	1	..
Oregon	..	..	..
California	1	3	11
Territories and Possessions			
Alaska	..	..	1
Hawaii	..	..	..
Puerto Rico	..	..	..
Total	7	15	22

FOREIGN

International Congress of the History of Medicine.—The 16th International Congress of the History of Medicine will take place at the School of Medicine of Montpellier (France) Sept. 22-28, under the general presidency of the dean of the faculty, Monsieur le doyen Giraud. Registrations will be accepted by Prof. Turchini, chairman of the committee of organization, Faculty of Medicine, Montpellier (Herault, France). Subjects on the agenda are:

The relations of the School of Montpellier with the medical institutions of other nations in course of the centuries.
The history and development of the hospital institutions.
Medical iconography in the seventeenth century.
The contribution of the new world to therapy.

Registration fees are 4000 francs for members of the International Society of the History of Medicine, 6000 francs for nonmembers, and 3500 francs for accompanying persons. The Comité d'orgaisation is now accepting titles of papers for presentation together with a short résumé.

Congress of Psychotherapy in Barcelona.—The fourth International Congress of Psychotherapy will be held Sept. 1-7, organized by the Sociedad Española de Medicina Psicosomatica y Psicoterapia, at the Palacio Nacional de Montjuich, Barcelona, Spain. The congress theme will be "Psychotherapy and Existential Analysis." Sections have been organized on cultural anthropology, the influence of oriental psychology, clinical psychology, psychodrama, hypnosis and relaxation methods, and other topics. Reports will be simultaneously translated into the four official languages: Spanish, English, French, and German. The program includes speakers from Switzerland, Cuba, Spain, the Netherlands, France, Austria, Germany, Israel, Peru, and the U. S. For information write the Secretary, International Congress of Psychotherapy, Cátedra de Psiquiatría de la Facultad de Medicina, Casanova, 143, Barcelona, Spain.

EXAMINATIONS AND LICENSURE

EDUCATIONAL COUNCIL FOR FOREIGN MEDICAL GRADUATES, INC.

Educational Council for Foreign Medical Graduates, Inc.: The American medical qualification examination to be given henceforth twice a year for foreign medical graduates. Medical Schools in the United States and Foreign Countries, Sept. 23. Final date for filing application is June 23. Executive Director, Dr. Dean F. Smiley, 1710 Orrington Ave., Evanston, Illinois.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: *Examination.* Montgomery, June 17-19. Sec., Dr. D. G. Gill, State Office Building, Montgomery 4.

ARIZONA:* *Examination and Reciprocity.* Phoenix, April 16-18. Exec. Sec., Mr. Robert Carpenter, 826 Security Bldg., Phoenix.

ARKANSAS:* *Examination.* Little Rock, June 5-6. Sec., Dr. Joe Verser, Harrisburg.

CALIFORNIA: *Written Examination.* San Francisco, June 16-19; Los Angeles, August 18-21; Sacramento, Oct. 20-23. *Oral Examination.* San Francisco, June 14; Los Angeles, August 16; San Francisco, November 15. *Oral and Clinical Examination for Foreign Medical School Graduates.* San Francisco, June 15; Los Angeles, August 17; San Francisco, November 16. Sec., Dr. Louis E. Jones, 1020 N Street, Sacramento.

COLORADO:* *Examination and Reciprocity.* Denver, June 10-11. Exec. Sec., Mrs. Beulah H. Hudgens, 715 Republic Bldg., Denver 2.

DELAWARE: *Examination and Reciprocity.* Dover, July 8-10. Sec., Dr. Joseph S. McDaniel, Professional Bldg., Dover.

FLORIDA:* *Examination.* Miami, June 29-July 1. Sec., Dr. Homer L. Pearson, 901 N. W. 17th St., Miami.

GEORGIA: *Examination and Reciprocity.* Atlanta and Augusta, June. Sec., Mr. C. L. Clifton, 224 State Capitol, Atlanta.

IDAHO: *Examination.* Boise, July 14-16. Exec. Sec., Mr. Armand L. Bird, 364 Sonna Bldg., Boise.

INDIANA: *Examination.* Indianapolis, June 18-20. Exec. Sec., Miss Ruth V. Kirk, 538 K. of P. Bldg., Indianapolis.

IOWA:* *Examination.* Iowa City, June 16-18. Exec. Sec., Mr. Ronald V. Saf, State Office Bldg., Des Moines 19.

KANSAS:* *Examination.* Kansas City, June 13-14. Sec., Dr. F. J. Nash, New Brotherhood Bldg., Kansas City.

LOUISIANA: *Examination and Reciprocity.* New Orleans, June 5-7. Sec., Dr. Edwin H. Lawson, 930 Hibernia Bank Bldg., New Orleans.

MARYLAND: *Examination.* Baltimore, June 17-20. Sec., Dr. Frank K. Morris, 1211 Cathedral St., Baltimore 1.

MASSACHUSETTS: *Examination.* Boston, July 15-18. Sec., Dr. Robert C. Cochrane, Room 37 State House, Boston.

MICHIGAN:* *Examination.* Ann Arbor and Detroit, June 9-11. Sec., Dr. E. C. Swanson, 118 Stevens T. Mason Bldg., West Michigan Ave., Lansing 8.

MINNESOTA:* *Examination.* Minneapolis, April 15-17. Sec., Dr. F. H. Magney, 230 Lowry Medical Arts Bldg., St. Paul 2.

MISSOURI: *Examination.* St. Louis, May 30-31. Ex. Sec., Mr. John A. Hailey, Jefferson City.

NEBRASKA:* *Examination.* Omaha, June. Sec., Mr. Husted K. Watson, Room 1009, State Capitol Bldg., Lincoln 9.

NEW MEXICO:* *Examination and Reciprocity.* Santa Fe, May 19-20. Sec., Dr. R. C. Derbyshire, 227 E. Palace Ave., Santa Fe.

NEW YORK: *Examination.* Albany, Buffalo, New York City and Syracuse, June 24-26. Sec., Dr. Stiles D. Ezell, 23 S. Pearl St., Albany.

NORTH CAROLINA: *Endorsement.* Asheville, May 5. *Examination.* Raleigh, June 16-19. *Endorsement.* Raleigh, June 17. Asst. Sec., Mrs. Louise J. McNeill, Professional Bldg., Raleigh.

NORTH DAKOTA: *Examination.* Grand Forks, July 9-11. *Reciprocity.* Grand Forks, July 12. Sec., Dr. C. J. Glaspel, Grafton.

OHIO: *Examination.* June 19-21. Sec., Dr. H. M. Platter, 21 West Broad St., Columbus 15.

OKLAHOMA:* *Examination.* Oklahoma City, June 3-4. Sec., Dr. E. F. Lester, 813 Braniff Bldg., Oklahoma City.

PENNSYLVANIA: *Examination.* Philadelphia and Pittsburgh, July 8-10. Acting Sec., Mrs. Marguerite G. Steiner, Box 911, Harrisburg.

SOUTH CAROLINA: *Reciprocity.* Myrtle Beach, May 13; *Examination.* Columbia, June 24-25. Sec., Dr. H. E. Jervey, 1329 Blanding St., Columbia.

SOUTH DAKOTA:* *Examination.* Rapid City, August 12-13. Exec. Sec., Mr. John C. Foster, 300 First National Bank Bldg., Sioux Falls.

TEXAS:* *Examination and Reciprocity.* Fort Worth, June 23-25. Sec., Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth 2.

UTAH: *Examination.* Salt Lake City, July 9-11. Director, Mr. Frank E. Lees, 324 State Capitol Bldg., Salt Lake City 1.

VIRGINIA: *Examination.* Richmond, June 12-14. *Reciprocity.* Richmond, June 11. Address: Board of Medical Examiners, 631 First St., S. W., Roanoke.

WASHINGTON:* *Examination.* Seattle, July 14-16. Administrator, Mr. Thomas A. Carter, Capitol Bldg., Olympia.

WEST VIRGINIA: *Examination and Reciprocity.* Charleston, July (date not set). Sec., Dr. N. H. Dyer, State Office Bldg., No. 5, Charleston.

WISCONSIN:* *Endorsement.* Madison, April 25. *Examination.* Milwaukee, July 8-10. Sec., Dr. Thomas W. Tormey, Jr., 1140 State Office Bldg., 1 West Wilson St., Madison.

WYOMING: *Examination and Reciprocity.* Cheyenne, June 2 and Oct. 6. Sec., Dr. Franklin D. Yoder, State Office Bldg., Cheyenne.

ALASKA:* On application in Anchorage and Juneau. Sec., Dr. W. M. Whitehead, 172 South Franklin St., Juneau.

GUAM: *Subject to Call.* Act. Sec., Dr. S. F. Provencher, Agana.

HAWAII: *Examination.* Honolulu, July 14-15. Sec., Dr. I. L. Tilden, 1029 Kapiolani St., Honolulu.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ALASKA: *Examination.* Juneau, Nov. 4. Sec., Dr. R. Harrison Leer, Room 204, Alaska Office Bldg., Juneau.

ARKANSAS: *Examination.* Little Rock, May 5-6. Sec., Mr. S. C. Dellinger, Zoology Department, University of Arkansas, Fayetteville.

COLORADO: *Examination and Endorsement.* Denver, May 7-8. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver 18.

CONNECTICUT: *Examination and Endorsement.* New Haven, June 14. Exec. Asst., Mrs. Regina G. Brown, 258 Bradley St., New Haven 10.

DISTRICT OF COLUMBIA: *Examination.* Washington, April 14-15. Deputy Director, Commission on Licensure, Mr. Paul Foley, 1740 Massachusetts Ave., N. W., Washington 6.

FLORIDA: *Examination.* Miami, June 7. Sec., Mr. M. W. Emmel, Box 340, Gainesville.

KANSAS: *Examination.* Kansas City, June 3-4. Sec., Dr. R. E. Stowell, University of Kansas School of Medicine, Kansas City 12.

NEW MEXICO: *Examination.* Santa Fe, April 20. *Reciprocity.* Santa Fe, March 26. Sec., Mrs. Marguerite Cantrell, Box 1522, Santa Fe.

SOUTH DAKOTA: *Examination.* Vermillion, June 6-7. Sec., Dr. Gregg M. Evans, 310 E. 15th St., Yankton.

RHODE ISLAND: *Endorsement.* Providence, April 30. *Examination.* Providence, May 14. Administrator of Professional Regulation, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

TEXAS: *Examination.* April 18-19, Austin, Galveston and Houston. Sec., Bro. Raphael Wilson, 407 Perry Brooks Bldg., Austin.

WISCONSIN: *Examination.* Milwaukee, June 7. Sec., Mr. William H. Barber, 621 Ransom St., Ripon.

*Basic Science Certificate required.

GOVERNMENT SERVICES

AIR FORCE

Space Medicine.—Academic rank as the world's first professor of space medicine has just been awarded to Dr. Hubertus Strughold at the Air Force School of Aviation Medicine. The school grants to teaching members of its staff the same academic status they would have at a comparable civilian institution. Dr. Strughold, who founded the school's department of space medicine 10 years ago, has been known until now as professor of aviation medicine. Renowned as the most eminent living authority on human flight in space, Dr. Strughold now serves as the school's advisor for research. He still lectures to student medical officers, however, on space medicine.

ARMY

New Medico-Dental Society in Italy.—The Southern European Task Force Medico-Dental Society opened a new point of contact for international exchange of medical knowledge on April 2, when 12 Italian physicians and dentists and SETAF medical and dental officers met at Camp Passalacqua, in Verona, to hear a talk by Col. H. Beecher Dierdorff on maxillofacial injuries. Colonel Dierdorff has been chief of oral surgery in several of America's largest Army hospitals. Capt. Kenneth Rowe is president of the society. Presiding at the meeting was Lieut. Col. George W. Grabiak, SETAF dental surgeon.

VETERANS ADMINISTRATION

Hospital News.—Dr. Oskar Diethelm, psychiatrist-in-chief, the New York Hospital (Payne Whitney Psychiatric Clinic), addressed the staff of the VA Hospital at Northport, N. Y., on March 25, on "The Concept of Regression in Schizophrenia."

PUBLIC HEALTH SERVICE

New Radiological Health Division.—Creation of a new division of radiological health in the Public Health Service was announced March 28 by Marion B. Folsom, secretary of the Department of Health, Education, and Welfare. The new division will provide technical assistance to state agencies in dealing with medical, industrial, and other activities involving public exposure to radiation, and it will also conduct research and training programs.

Ten years ago, the PHS radiation unit consisted of one physician and a secretary. Today, 51 persons and a budget of almost $400,000 are involved in radiation work. Under the new division, it is expected the program will expand to include a staff of 76 persons, with a budget of over $600,000, in fiscal year 1959. Chief of the new division will be Dr. Francis J. Weber, a career officer in the PHS and at present medical director of the department's Denver regional office, covering Colorado, Utah, Wyoming, Montana, and Idaho. The surgeon general recently announced the appointment of a National Advisory Committee on Radiation which, under the chairmanship of Dr. Russell H. Morgan, professor of radiology, Johns Hopkins University School of Medicine, held its first meeting on March 13.

Poliomyelitis Vaccination Urged.—Surgeon General Leroy E. Burney announced that revised estimates indicate that 48,500,000 persons in the susceptible age group, under 40 years of age, have not been vaccinated against poliomyelitis. The Salk vaccine was an important factor in the 85% reduction of paralytic cases over the past two years, but poliomyelitis comes in cycles. Even in prevaccine years, there have been periods of dramatic reductions. Epidemic years have always followed. Should the attack rate in 1958 prove to be comparable to that of 1952, 30,000 cases of poliomyelitis, a substantial number of them paralytic, could occur among those who have not been vaccinated. This potential toll of suffering and lifelong disability is unnecessary. The way to prevent it is simple: get vaccinated, and start now. Parents of pre-school-age children run a special risk because the attack rate of the disease is high among small children. If these children have been vaccinated, they themselves are protected against paralysis, but they can still harbor the virus and pass it on to their parents.

Personal.—Dr. Robert M. Farrier has been appointed assistant director of the National Institutes of Health Clinical Center, Bethesda, Md., succeeding Dr. Stuart M. Sessoms, now assistant director of the National Cancer Institute. Since 1954, Dr. Farrier has been on special assignments for the headquarters office of the Bureau of Medical Services of the Public Health Service.

NATIONAL SCIENCE FOUNDATION

Personal.—Arthur W. Martin, Ph.D., has been named program director for regulatory biology, National Science Foundation. He is on leave of absence from the University of Washington where he is chairman, department of zoology. During the current fiscal year, the foundation expects to award grants totaling more than a million dollars for basic research in this field, primarily to educational institutions in the United States. Dr. Martin has done research in cellular metabolism, muscle atrophy in mammals, and pharmacological actions of barbiturates and their antagonists.

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DEATHS

Abbott, Melvin Dewey ✱ Captain, U. S. Navy, retired, Pembroke, Ga.; born in Adrian, Mich., Nov. 6, 1899; Detroit College of Medicine and Surgery, 1926; appointed assistant surgeon in the medical corps of the U. S. Navy with the rank of lieutenant (jg) in June, 1925; assigned to the U. S. Naval Hospital, New York City, for internship; later served at the Naval Training Station, Hampton Roads, Va., at four Naval hospitals, on eight U. S. ships, and at various other Naval shore stations; attained the rank of lieutenant in 1930, lieutenant commander in 1938, commander in 1942, and captain, 1943; in 1954 while serving at the Navy Recruiting Station in Macon, was placed on the retired list of Naval officers after completing more than 29 years of active service; service member of the American Medical Association; fellow of the American College of Surgeons; served a residency at the Flower and Fifth Avenue Hospitals in New York City; died in the Bulloch County Hospital, Statesboro, Feb. 17, aged 58, of a wound received when shot by a caretaker.

Alexander, William Cobb, Clifton Hill, Mo.; Louisville (Ky.) and Hospital Medical College, 1908; died in the Woodland Hospital, Moberly, Feb. 15, aged 73.

Baker, Edwin Wilson, Fremont, Ohio; University of Buffalo School of Medicine, 1883; on the staff of the Memorial Hospital; died Feb. 21, aged 98, of heart disease and arteriosclerosis.

Ballou, De Forrest Jr., Linwood, N. J.; University of Pennsylvania School of Medicine, Philadelphia, 1913; served in the regular Army; veteran of World Wars I and II; formerly on the staff of the Veterans Administration Hospital in Castle Point, N. Y.; died in the Shore Memorial Hospital, Atlantic City, N. J., Feb. 13, aged 72.

Bentley, Frederick W., North Tonawanda, N. Y.; New York Homeopathic Medical College and Hospital, New York City, 1894; died in the DeGraff Memorial Hospital Feb. 23, aged 87, of gastric carcinoma and hepatic metastases.

Bicknell, George C. ✱ Indian Head, Md.; Baltimore Medical College, 1904; associated with Physicians Memorial Hospital in La Plata; died in Prince Frederick Feb. 10, aged 77.

Blackmore, Thomas Albert ✱ Windsor, Mo.; Missouri Medical College, St. Louis, 1898; died Jan. 26, aged 85, of injuries received in an automobile accident.

✱ Indicates Member of the American Medical Association.

Boden, Leo Meyer ✱ Buffalo; Albert-Ludwigs-Universität Medizinische Fakultät, Freiburg, Baden, Germany, 1925; member of the American Academy of General Practice; died Feb. 13, aged 59, of heart disease.

Bond, Theron Blain, Geneva, N. Y.; University of Buffalo School of Medicine, 1912; member of the Medical Society of the State of New York; veteran of World War I; died in the Veterans Administration Hospital, Canandaigua, Jan. 30, aged 74.

Brown, Swan Wilmer, Cadillac, Mich.; Jefferson Medical College of Philadelphia, 1900; died in the Mercy Hospital Feb. 26, aged 91, of coronary occlusion.

Brown, Walter Earl ✱ Pittsburgh; Jefferson Medical College of Philadelphia, 1911; on the staff of the Homestead (Pa.) Hospital, where he died Feb. 12, aged 72, of arteriosclerotic heart disease.

Buchheit, Rudolph George ✱ Buffalo; University of Buffalo School of Medicine, 1928; member of the Association for Research in Ophthalmology; associated with Our Lady of Victory Hospital in Lackawanna and the Mercy Hospital; died Feb. 18, aged 56.

Burns, Francis John ✱ Spokane, Wash.; Loyola University School of Medicine, Chicago, 1921; county coroner; veteran of World Wars I and II; associated with Deaconess and St. Luke's hospitals; died in the Sacred Heart Hospital Feb. 4, aged 65, of carcinoma of the lung.

Code, John Nicholas, Brightwaters, N. Y.; University College, Dublin, Ireland, 1944; member of the American Society of Anesthesiologists and the Medical Society of the State of New York; on the staff of the Southside Hospital and the Lakeside Hospital, where he died Feb. 20, aged 45, of a heart attack.

Conant, Edgar Francis ✱ Denver; Columbian University Medical Department, Washington, D. C., 1894; died Feb. 7, aged 80, of coronary occlusion.

Cross, Frank Bethel ✱ Brooklyn; born in 1879; Columbia University College of Physicians and Surgeons, New York City, 1900; clinical professor emeritus of medicine at State University of New York College of Medicine; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; past-president of the Brooklyn Society of Internal Medicine; emeritus chief of the medical staff and member of

the board of managers, Methodist Hospital; consulting physician at St. John's Episcopal, Victory Memorial, Midwood, Bay Ridge, and Samaritan hospitals all in Brooklyn, St. Luke's Hospital in Newburgh, and John T. Mather Memorial Hospital in Port Jefferson, N. Y.; died in the Louden-Knickerbocker Hall, Amityville, N. Y., Feb. 26, aged 78, of coronary occlusion.

DeBonis, Domenico A. ● West Hartford, Conn. (licensed in Connecticut in 1893); died in St. Francis Hospital, Hartford, Feb. 17, aged 93, of bronchopneumonia and arteriosclerotic heart disease.

Donehoo, J. Frank, Jackson, Miss.; University of Pennsylvania Department of Medicine, Philadelphia, 1893; an associate member of the American Medical Association; for many years practiced in Washington, Pa., where he served on the staff of the Washington Hospital; died Feb. 8, aged 88.

Donnelly, James Gordon ● San Francisco; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1912; service member of the American Medical Association; veteran of World Wars I and II; for many years manager of the Veterans Administration Hospital, Fort Miley; died in St. Mary's Hospital Feb. 18, aged 67, of occlusive coronary disease.

Draper, William Banford, Denver; McGill University Faculty of Medicine, Montreal, Que., Canada, 1924; professor of pharmacology at the University of Colorado School of Medicine; served in the Canadian Army during World War I; died in Tucson, Ariz., Feb. 18, aged 64, of cerebral hemorrhage.

Ewart, Thomas Alexander, Long Beach, Calif.; Michigan College of Medicine and Surgery, Detroit, 1901; formerly associated with the Indian Service; on the staffs of the Seaside Memorial, St. Mary's, and Community hospitals; died Feb. 18, aged 84, of congestive heart disease and arteriosclerosis.

Fordham, Henry Clendenin, Chapel Hill, N. C.; Harvard Medical School, Boston, 1953; service member of the American Medical Association; certified by the National Board of Medical Examiners; served in the medical corps of the U. S. Naval Reserve; resident, North Carolina Memorial Hospital; died Feb. 13, aged 28, of encephalitis.

Fowzer, Lloyd Robert, Milwaukee; College of Medicine and Surgery (**Physico Medical**) Chicago 1911; died Feb. 16, aged 68.

Furman, Richard B. ● Sumter, S. C.; Medical College of the State of South Carolina, Charleston, 1889; died in the Twomey Hospital Feb. 22, aged 91, of arteriosclerotic heart disease, bronchopneumonia and acute gastroenteritis.

Goldfain, Samuel Joseph ● Baton Rouge, La.; George Washington University School of Medicine, Washington, D. C., 1922; fellow of the American College of Surgeons; veteran of World War I; on the staffs of the Baton Rouge General Hospital and Our Lady of the Lake Hospital; died in Jackson Jan. 17, aged 58.

Haeberlin, John Benjamin ● Chicago; Northwestern University Medical School, Chicago, 1900; fellow of the American College of Surgeons; at one time associate clinical professor of surgery at Loyola University School of Medicine; served on the staff of St. Bernard's Hospital; died Feb. 12, aged 81, of coronary insufficiency.

Hagen, Beatrice A. Todd, Zanesville, Ohio; University of Buffalo School of Medicine, 1900; served as secretary of the Muskingum County Academy of Medicine; for many years health commissioner of Muskingum County; died Feb. 13, aged 81.

Hannings, Herschel Victor, Martin, Tenn.; University of Tennessee Medical Department, Nashville, 1908; died in the Obion County Hospital in Union City, Dec. 26, aged 73.

Harvey, John King ● Salina, Kan.; University of Kansas School of Medicine, Kansas City, Kan., 1908; associated with Asbury Hospital and St. John's Hospital, where he died Feb. 20, aged 76, of cerebrovascular occlusion.

Hendrickson, Clifford Wood, Bethel, Conn.; Long Island College Hospital, Brooklyn, 1901; an associate member of the American Medical Association; formerly practiced in New Rochelle, N. Y.; veteran of World War I; died in Danbury (Conn.) Hospital, Jan. 30, aged 79, of bronchopneumonia, following a fracture, and duodenal ulcer.

Holderith, Charles Peter ● New Orleans; Medical Department of Tulane University of Louisiana, New Orleans, 1906; veteran of World War I; served as state representative; died Feb. 22, aged 76.

Hyer, Yeadon Mazyck, Darlington, S. C.; Medical College of South Carolina, Charleston, 1933; veteran of World War II; formerly health officer of Chester County, Hampton and Allendale Counties; associated with McLeod Infirmary in Florence, where he died Jan. 29, aged 50, of pneumonia.

Jenson, William Andrew ● Chicago; Chicago College of Medicine and Surgery, 1912; associated with the Swedish Covenant Hospital, where he died Feb. 26, aged 78.

Johnson, Francis Bonneau ● Charleston, S. C.; Medical College of the State of South Carolina, Charleston, 1903; specialist certified by the American Board of Pathology; fellow of the American College of Physicians; professor emeritus of clinical

pathology at his alma mater; past-president of the South Carolina Medical Association and the Tri-State Medical Association of the Carolinas and Virginia; died Jan. 16, aged 76, of carcinoma of the prostate.

Johnson, Julius • Minneapolis; Minneapolis College Physicians and Surgeons, medical department of Hamline University, 1906; member of the American Psychiatric Association; served on the staffs of Swedish, St. Barnabas, Fairview, St. Andrew's, Deaconess, and Glenwood Hills hospitals; died Feb. 8, aged 80, of myocardial infarction.

Johnson, S. Lloyd • Catonsville, Md.; Johns Hopkins University School of Medicine, Baltimore, 1918; member of the American Academy of General Practice; first president of the Maryland Academy of General Practice; formerly on the faculty of the University of Maryland School of Medicine and College of Physicians and Surgeons; veteran of World War I; died in the Johns Hopkins Hospital, Baltimore, Feb. 4, aged 67, of acute leukemia.

Jordan, Saunders Robert, Carolina Beach, N. C.; Medical College of Virginia, Richmond, 1899; veteran of World War I; died Feb. 6, aged 84.

Joyce, Thomas Francis, Los Angeles; Bellevue Hospital Medical College, New York City, 1898; an associate member of the American Medical Association; served as medical superintendent of the Pacific Colony–State Narcotic Hospital in Spadra, Calif.; died Feb. 10, aged 84, of bilateral bronchopneumonia.

Kirby, Darwin • Champaign, Ill.; Northwestern University Medical School, Chicago, 1907; member of the Industrial Medical Association; fellow of the American College of Surgeons; past-president of the American Association of Railroad Surgeons; died in the Norbury Sanitarium in Jacksonville Feb. 15, aged 74, of coronary occlusion and general and cerebral arteriosclerosis.

Kotz, Jacob, Washington, D. C.; born Dec. 25, 1895; George Washington University School of Medicine, Washington, D. C., 1917; formerly clinical professor of obstetrics and gynecology at George Washington University School of Medicine; specialist certified by the American Board of Obstetrics and Gynecology; an associate member of the American Medical Association; fellow of the International College of Surgeons and the American College of Surgeons; veteran of World War I; served on the staffs of the George Washington, Sibley Memorial, and Prince Georges hospitals and Doctors Hospital, where he died Feb. 28, aged 62, of arteriosclerotic heart disease.

Landis, Robert K. • Nashville, Tenn.; (licensed in Tennessee in 1909); member of the American Academy of General Practice; on the staffs of the Nashville General Hospital and the Baptist Hospital, where he died Feb. 2, aged 73, of acute myocardial infarction.

Leonard, Ward Holmes • Kansas City, Mo.; University of Pennsylvania Department of Medicine, Philadelphia, 1897; veteran of World War I; on the staff of the Trinity Hospital, where he died Jan. 30, aged 86, of myocardial infarction.

Lewis, George Everie • Lincoln, Neb.; University of Nebraska College of Medicine, Omaha, 1925; fellow of the International College of Surgeons; veteran of World War I; associated with St. Elizabeth's and Bryan Memorial hospitals; died in the Veterans Administration Hospital, Omaha, Feb. 7, aged 60, of cerebral thrombosis.

Lightner, Homer C., Ideal, Ga.; Atlanta College of Physicians and Surgeons, 1906; veteran of World War I; formerly associated with the Veterans Administration in Macon; died in Montezuma Feb. 8, aged 77, of a heart attack.

Linsey, Philip Robert • Cleveland; University of Wooster Medical Department, Cleveland, 1910; member of the American Academy of General Practice; associated with the Doctors Hospital in Cleveland Heights and Polyclinic Hospital; died Feb. 10, aged 71.

Logan, Jesse Robert • Evansville, Ind.; Indiana University School of Medicine, Indianapolis, 1927; veteran of World War II; member of the American Academy of General Practice; on the staffs of the Deaconess and St. Mary's hospitals; died Feb. 16, aged 57, of a malignant brain tumor.

Long, Felix Benjamin • Starkville, Miss.; University of Tennessee College of Medicine, Memphis, 1914; associated with the Felix Long Memorial Hospital; died in the Price and Black Hospital in Lamesa Feb. 6, aged 75, of coronary occlusion.

Lorenz, William Frederick • Madison, Wis.; born in New York City Feb. 15, 1882; University and Bellevue Hospital Medical College, New York City, 1903; emeritus professor of neuropsychiatry at the University of Wisconsin Medical School; specialist certified by the American Board of Psychiatry and Neurology; member of the American Psychiatric Association and the Central Neuropsychiatric Association; fellow of the American College of Physicians; veteran of the Spanish–American War; served during World War I and received the Distinguished Service Medal; engaged as special expert in research investigation of pellagra for the U. S. Public Health Service, 1914-1915; past-president of the Wisconsin State Board of Control, Madison Police and Fire Commission, and the Wisconsin Rehabilitation Board; formerly director of

the Wisconsin Psychiatric Institute and president of the staff of St. Mary's Hospital; died Feb. 18, aged 76, of heart disease.

Love, Luther Samuel ✱ Nashville, Tenn.; University of Tennessee Medical Department, Nashville, 1909; associated with the Central State Hospital; died Feb. 10, aged 76.

Love, Samuel Glenn, Asheville, N. C.; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1914; died Feb. 14, aged 66.

McCool, Joseph Letelle, Monrovia, Calif.; University of Pennsylvania Department of Medicine, Philadelphia, 1900; an associate member of the American Medical Association; specialist certified by the American Board of Ophthalmology; member of the American Academy of Ophthalmology and Otolaryngology; formerly on the faculty of the University of California School of Medicine; at one time practiced in Philadelphia and Portland, Ore.; died Feb. 12, aged 78, of embolism.

McCulloch, John Max ✱ Maryville, Tenn.; University of Tennessee College of Medicine, Memphis, 1935; on the staff of the Blount Memorial Hospital, where he died Feb. 6, aged 51, of acute posterior myocardial infarction.

McElhinney, John Samuel, Buffalo, Mo.; Eclectic Medical University, Kansas City, Mo., 1906; died in Lebanon Dec. 16, aged 91.

McGuire, Bennet Joseph ✱ Pittston, Pa.; University of Pittsburgh School of Medicine, 1925; formerly an officer in the medical corps of the U. S. Army Reserve; member of the Radiological Society of North America; past-vice-president of the Pennsylvania Radiological Society; for many years member of the board of health of West Pittston; associated with Mercy Hospital in Wilkes-Barre and the Pittston Hospital; vice-president and for many years member of the board of managers of Luzerne County Industrial School at Kis-Lyn; died in the Veterans Administration Hospital, Wilkes-Barre, Feb. 18, aged 62, of cancer.

Mair, William Lewis, Detroit; McGill University Faculty of Medicine, Montreal, Que., Canada, 1906; died in the Public Hospital, Clinton, Ont., Canada, Feb. 11, aged 76, of cerebral thrombosis.

Margolin, Berthe ✱ Tulsa, Okla.; Fort Worth School of Medicine, Medical Department of Texas Christian University, 1917; served on the staff of St. John's Hospital; died Feb. 1, aged 65, of injuries received when struck by her automobile.

Meek, Marcus L., Abington, Ind.; Central College of Physicians and Surgeons, Indianapolis, 1895; died in the Reid Memorial Hospital, Richmond, Feb. 10, aged 101, of arteriosclerotic heart disease.

Mendel, James Harold ✱ Miami, Fla.; born in Wheeling, W. Va., Aug. 17, 1894; Jefferson Medical College of Philadelphia, 1918; served on the faculty of his alma mater; at one time practiced in Philadelphia, where he was associated with Jefferson Hospital, Germantown Dispensary and Hospital, and the Memorial Hospital; specialist certified by the American Board of Otolaryngology; in 1935 was awarded a Bronze Medal by the American Medical Association for his exhibit on ear drums and their interpretation; member of the American Academy of Ophthalmology and Otolaryngology; on the staff of the Doctors' Hospital in Coral Gables; for many years on the staff of the Mercy Hospital; died in Coral Gables, Feb. 5, aged 63, of myocardial infarction.

Miller, Robert Carlysle, Gastonia, N. C.; North Carolina Medical College, Charlotte, 1909; member of the Medical Society of the State of North Carolina; past-president of the Gaston County Medical Society; associated with Gaston Memorial, Garrison General, and Gaston County Negro hospitals; died Feb. 4, aged 72, of cerebral hemorrhage.

Mixsell, Harold Ruckman ✱ New York City; Columbia University College of Physicians and Surgeons, New York City, 1910; served on the faculty of the University and Bellevue Hospital Medical College; specialist certified by the American Board of Pediatrics; member of the American Academy of Pediatrics; veteran of World War I; consulting pediatrician, Community Hospital in Glen Cove, St. Luke's, Woman's, and Beekman-Downtown hospitals; formerly editor of the *Archives of Pediatrics;* died in the New York Hospital Feb. 13, aged 72.

Moore, John Frederick ✱ Nashville, Tenn.; George Washington University School of Medicine, Washington, D. C., 1927; retired from the Veterans Administration Aug. 11, 1952; died Feb. 9, aged 58, of cerebral thrombosis.

Moore, John Milton, Chicago; McGill University Faculty of Medicine, Montreal, Que., Canada, 1893; died Feb. 2, aged 89, of arteriosclerotic heart disease, generalized arteriosclerosis, and hypertension.

Mullen, James Emmett ✱ Toledo, Ohio; Loyola University School of Medicine, Chicago, 1936; member of the Industrial Medical Association; fellow of the American College of Surgeons; associated with St. Charles and Mercy hospitals; died Feb. 8, aged 49, of a heart attack.

Zahn, Benjamin, J., Oak Park, Ill.; Chicago Homeopathic Medical College, 1900; died in the Oak Park Hospital Feb. 10, aged 89, of encephalomalacia and cerebral arteriosclerosis, general arteriosclerosis, and arteriosclerotic heart disease.

FOREIGN LETTERS

AUSTRIA

Post-traumatic Subdural Effusions.—At the meeting of the Society of Physicians in Vienna on Feb. 7, Dr. K. Holub said that colorless or yellowish subdural effusions (hygroma) produce symptoms resembling those of subdural hematoma. In less severe cases the patients complain of headache, vertigo, and unsteady gait but show no neurological findings. These patients are often treated on the basis of an erroneous diagnosis such as post-traumatic neurosis or compensation neurosis. It is important to carry out all the necessary diagnostic examinations, since an operation performed in time often cures these patients.

Passage of Poliomyelitis Virus Through Filters.—At the same meeting Drs. W. Auerswald and J. Klima said that accidents associated with poliomyelitis vaccine in 1955 were due to the presence of virus aggregates in the vaccine. Previously Syverton had demonstrated the infectivity of the tissue culture noncytopathogenic vaccine in cortisone-treated monkeys, and Taylor observed in the vaccine sediment after ultracentrifugation aggregates of virus. Therefore a second filtration of the single-strain virus pools three days after complete inactivation was prescribed as a minimum requirement. It became evident, however, that filtration induced loss of antigenicity. Studies on the behavior of polioviruses during filtration were formerly made on active virus which could be titrated on the tissue culture, but it is doubtful that results obtained with active virus can be compared with those of the formaldehyde-treated virus interacting with filters. The author therefore modified the method Sharp used with influenza virus to evaluate directly the particle numbers of the virus suspension before and after filtration. The virus suspension is ultracentrifuged at 26,000 rpm in an analytical cell, quantitatively on the agar-coated bottom. After fixation the virus is transferred to a collodion replica and the particle number is evaluated in the electron microscope. By means of controls with the supernatant fluid it was shown that all of the virus had been recovered. In this way the selective properties of a filter can be evaluated in terms of lost particles. In these experiments it was further possible to obtain pictures of aggregates of poliovirus contained in the virus suspension before filtration while the same suspension after filtration was free from aggregates. This way of direct observation may improve control measures during vaccine production and enhance the safety and antigenicity of the finished product.

Epithelioid Cell Reaction.—At a meeting of the Society of Physicians on Jan. 31, Dr. St. Wuketich reported on histological examinations made on 489 patients who were operated on for carcinoma. The tissues in 29 of these revealed an epithelioid cell reaction of the regional lymph nodes. This reaction was demonstrated in the lymph nodes with and without cancer. The two main types of epithelioid cell reactions include the small foci of epithelioid cell proliferation and the granulomatous epithelioid cell proliferation with extensive foci. The inflammatory changes of the lymph node in an epithelioid cell proliferation with a small focus is almost always associated with the presence of reticulosis of the sinuses and with an increased activity of the germ centers and are similar to those seen in patients with subacute cervical and nuchal lymphadenitis. The picture of the epithelioid cell proliferation could not be differentiated with certainty from the epithelioid tuberculosis and Boeck's sarcoid. The epithelioid cell lymph node reaction is particularly present in patients with regressive carcinomatous changes after x-ray treatment and is initiated by the disintegration products of the tissues of the tumor.

Pelvic Lymph Nodes.—At the meeting of the society of Gynecologists and Obstetricians in January in Vienna, Dr. G. Reiffenstuhl said that the efferent lymph vessels and the lymph nodes of the pelvis were made visible in cadavers of newborn girls by the injection of a dye, and after an adequate fixation were examined under a dissecting microscope. Because the nomenclature of the pelvic lymph nodes is not uniform and has been changed repeatedly, the proposal was made to name the nodes according to their position with respect to the blood vessels, so that their names would designate their approximate topography. The regional lymph nodes of the uterine cervix are situated along and behind the large parietal blood vessels, starting at the infrapiriform foramen and extending to the aorta. The pubogluteal lymph nodes are situated behind the large blood vessels in the area of the spina ischiadica and can be reached only with difficulty. These studies revealed that the lymphatic system of the pelvis consists of a widespread

The items in these letters are contributed by regular correspondents in the various foreign countries.

network. Because of the number of regional lymph nodes, cervical cancers may metastasize through a great variety of pathways.

Cancer of the Uterine Cervix.—At the same meeting Dr. H. Knaus said that the high death rate from carcinoma of the uterine cervix presented a complex problem. Although the operative mortality has decreased, two serious problems remain, namely, postoperative urethral fistula and recurrence in the regional lymph nodes. These complications may be prevented, however, by using the best operative technique. In the speaker's series of 117 radical operations (85 with Wertheim's and 32 with Schauta's method) only one woman died of a postoperative collapse of the lung. After a Wertheim's operation, a urethral fistula occurred in only one patient. In this patient exposure of the urethra in an already carcinomatous environment proved very difficult. This fistula was treated three months after the radical operation by implantation into the bladder, and the patient was cured of the fistula and carcinoma. The operation was performed in 1950 and the patient is still alive and well. The radical operation, including removal of the regional lymph nodes in 39 patients, revealed involvement of the lymph nodes in 19. Of the 116 patients who survived the operation, 24 died later (17 after Wertheim's and 7 after Schauta's operation). Of 55 who were operated on in the years from 1950 to 1953 (36 using Wertheim's and 19 using Schauta's technique) 40 appear to be permanently cured (25 after Wertheim's and 15 after Schauta's operation).

BRAZIL

Leprosy and Tuberculosis.—The results of the tuberculin and lepromin tests, carried out in preventoriums among children of persons with leprosy who consequently had been exposed to this disease lead Dr. N. S. Campos and co-workers (*International Journal of Leprosy*) to conclude that a state of specific resistance or immunity to tuberculosis due to leprous infection and a state of cross resistance due to tuberculous infection exist. Some of these leprosy contacts were lepromin-positive and had specific resistance, cross resistance, or both. They might appear to be healthy, or they might have lesions of the tuberculoid type of the disease. Others were lepromin-negative. They too might appear to be healthy, or they might have lesions of the indeterminate or lepromatous forms of the disease. The incidence of leprosy among close contacts was found to be much higher than is indicated by current epidemiological data. Correlation of the tuberculin and lepromin reactions led to an orientation of the criteria of surveillance of these children in preventoriums. Most attention should be given, first, to the lepromin-negative and tuberculin-positive children, and, next, to those who are negative to both lepromin and tuberculin. The high frequency of positive lepromin reactions in those without evident manifestations of leprosy and the high incidence of tuberculoid leprosy (nodular and sarcoidal) among the close contacts of patients with open forms of the disease suggested the existence of the lymph node complex without clinical manifestations, and that those manifestations, by their own characteristics, corresponded to the initial or inoculation lesion.

Blood Groups and Gastric Lesions.—Leite and Goffi (*Revista paulista de medicina*, vol. 52, January 1958) found the following blood group distribution in 124 patients with gastroduodenal ulcer, 98 with gastric carcinoma, and 1,818 unselected blood donors:

Blood Group	Gastric Carcinoma %	Peptic Ulcer, %	Unselected Blood Donors, %
A	55.0	35.5	38.7
B	5.1	9.7	10.1
O	34.8	49.1	47.7
AB	5.1	5.7	3.5

The authors concluded that there was a significant increase in the frequency of group A and a decrease of group O among patients with gastric carcinoma, and that there was no significant difference in the distribution of ABO blood groups among patients with peptic ulcer. This could be explained either through carcinogenic action by gene A, or through a protective action by gene O.

Mega-esophagus and Trypanosomiasis.—Rezende and Rassi (*O Hospital*, vol. 53, January, 1958) found that the endemic mega-esophagus and mega-colon which occur in certain areas of Brazil are definitely caused by Trypanosoma cruzi. The complement fixation test for Brazilian trypanosomiasis was positive in 89% of 170 unselected patients with mega-esophagus and in 68.6% of 277 with heart disease believed to be infected with T. cruzi. In 126 unselected patients with mega-esophagus, 50% showed signs of heart disease. Among 175 patients with chronic trypanosomal cardiopathy, 27.9% showed evidence of esophageal involvement. The authors concluded that the esophagus, when affected by trypanosomiasis, reveals the diagnosis much earlier than the heart. The proportion of males to females was 1.7:1 among those with trypanosomal cardiopathy; 2:1 among those with mega-esophagus; and 2.3:1 among those with associated cardiopathy and mega-esophagus.

Carcinoma of Uterine Cervix.—J. C. G. da Silveira (*Revista de gynecologica e d'obstetricia*, vol. 52, January, 1958) lists the following situations in the diagnosis of noninvasive carcinoma of uterine cervix in which conservative treatment could permit the extension of the lesions: (1) endocervical ascension or glandular penetration; (2) ectocervical

extension of carcinoma in situ beyond the habitual limits of vaginal section in the amputation or the conization of the cervix; (3) coexistence of an invasive carcinoma below the area of carcinoma in situ; and (4) precocious lymphatic metastasis of a superficial, apparently intraepithelial lesion. In any of these situations, pure excision of the cervix or total hysterectomy, conservative of ovaries and vagina, could result in disaster. In such an operation there is always the possibility of leaving areas of carcinoma in situ which could become invasive or of overlooking the present invasion or the precocious ganglional metastasis that would require radical hysterectomy with lymphadenectomy.

FINLAND

Adiposis Dolorosa.—According to Kirpila and Ripatti (*Nordisk medicin*, March 6, 1958) adiposis dolorosa is fairly common. In most of their series of 27 women with this disease, the fatty tumors had dominated the clinical picture by virtue of the pain they provoked, and in several cases they were the patients' sole complaint, although the patients had originally been sent to hospital as rheumatics. All of these women were very fat, their weights ranging from 57 to 92 kg. (125 to 202 lb.). Their ages ranged from 41 to 56 years. Their concomitant articular rheumatism was usually mild. The sedimentation rate was only slightly raised, the mean being lower for these patients than for rheumatics in general. Though a lookout was kept for hormonal disturbances, with special reference to the thyroid, there was only one case of thyroid adenoma.

Laboratory tests did not confirm the clinical impression of thyroid deficiency in two patients. Several patients showed well-marked neurasthenia. The tumors often showed a symmetrical distribution, being found on the inner side of the knee in many cases in the form of painful infiltrations of fat. Five patients also had such tumors above and below the knees. The buttocks and lumbar regions were also involved in several patients. The size of the tumors ranged from that of a cherry to that of a clenched fist. Adiposis dolorosa should not be diagnosed when no replica can be found elsewhere to single painful infiltration. All the patients in this series were given restricted diets and a thyroid preparation, but, since the cause of the disease is still unknown, it is not surprising that the treatment was often disappointing. Roentgen treatment gave some relief, and so did the removal of small and strictly limited tumors in three patients.

Myocardial Infarction.—Between 1952 and 1956 a hospital in Abo discharged as convalescents 370 patients who had been treated for acute cardiac infarction. Their subsequent fate from six months to five years after discharge was reported by Dr. E. Iisalo and co-workers (*Nordisk medicin*, Feb. 13, 1958). Of the 87 patients who died since discharge, death was due to some cardiovascular disease in 81 and was sudden in 76 of these. Five deaths were due to apoplexy. There were 41 patients who were not traced. The remaining 242 patients formed the basis of the study. Of these, 95 (40%) were unable to resume their previous occupations. The remaining 60% had returned to regular work from three to six months after discharge. The high rate of invalidism compares unfavorably with the corresponding rate reported by earlier observers and may reflect the comparatively advanced age of the Finnish patients. In 54 cases an electrocardiogram characteristic of cardiac infarction had become normal since discharge. They included 35 patients who were fit for work on reexamination. This high recovery rate after an alarming electrocardiogram at the time of an acute infarction suggests that this test may often fail to reveal the dynamic capacity of the heart to react to the demands made on it. Many in need of digitalis treatment did not receive it, and the follow-up examination showed that it had often not even approximately achieved the results to be expected of it when it was given.

MEXICO

El Medico.—In April Mexico's largest independent medical publication, *El Medico*, celebrated its seventh anniversary. All of Mexico's approximately 15,000 physicians receive this journal monthly. In addition to the Mexican issue, the editor prepares special limited editions for Venezuela, Columbia, Ecuador, and all Central American countries. This medical periodical comes closest to THE JOURNAL of the American Medical Association but since Mexico has no official professional organization, *El Medico* fights for economic, scientific, cultural, and social benefits for physicians. Recent issues to all countries have reprinted the new A. M. A. Code of Ethics for the benefit of 27,000 Latin American physicians. One newly developed section of *El Medico* is the section on practical medicine. Here little known technical procedures such as blood typing are discussed.

UNITED KINGDOM

Unidentified Respiratory Disease of Children.—An outbreak of respiratory disease, the cause of which is unknown, occurred in London and Birmingham. It was not due to any type of known influenza virus, and laboratory investigations which are proceeding have so far failed to find a cause. The Princess Louise Kensington Hospital for Children, London, recently admitted 104 such patients, and although at the time there was an increased incidence of

respiratory illness in London, the patients in this outbreak differed with respect to severity of illness and clinical features from those seen in other parts of the city. There was a history of rhinorrhea and sneezing for three to five days, sometimes with a cough, followed by noisy breathing and an increased respiratory rate (between 40 and 96 per minute, and in some up to 140 per minute). The children were pale and seemed to be in shock. Those under three months old were often cyanosed, with tachycardia, with pulse rate of 190 per minute. The breath sounds were harsh, and fine widespread rales developed later. A white frothy secretion was present in the mouth and pharynx, often requiring aspiration. Older children had a temperature varying from 100 to 105 F. (37.8 to 40.6 C), but younger children were afebrile.

The total leukocyte count varied from 6,000 to 15,000 with no significant disturbance of the ratio of polymorphonuclear cells to lymphocytes. No specific pathogens were isolated from the nose or throat. The sedimentation rate was raised in half the patients. Chest roentgenograms showed increased density, particularly in the lower lobes, and in a high proportion there was evidence of collapse-consolidation. Radiologic follow-up showed that these lesions resolved fairly rapidly. Half the patients required treatment in an oxygen tent, with an oxygen content of 80% and a humidity of 70 to 80%. Six children required digitalization for heart failure. Antibiotics and sulfonamides were given, but they were of little value, except in the prevention of secondary infection. Once the acute stage of respiratory infection was over, recovery was rapid. Two children died, however, from the illness.

Hypoglycemic Sulfonylureas.—There is still speculation on the precise mode of action of tolbutamide and carbutamide. Weaver and co-workers (*Brit. M. J.* 1:425, 1958) made investigations suggesting that tolbutamide does not act by increasing the effective level of insulin in the blood, as some observers have supposed. They examined the arteriovenous (A-V) glucose difference after an oral dose of 3 Gm. of tolbutamide in 10 patients with diabetes of recent onset not controlled by diet. A simultaneous estimation of the insulin-like activity of the serums was also made using the rat diaphragm technique before and after the administration of the drug. In addition studies were made to determine the effect of tolbutamide on the rate of disappearance of insulin labeled with I^{131} from the plasma of normal rabbits. The A-V glucose difference is an index of peripheral glucose uptake, if the peripheral blood flow remains unchanged and the arterial glucose level is constant. It was assumed that they remained so in these patients. Since no change in the A-V glucose difference was found this was taken as evidence that tolbutamide does not increase the uptake of glucose by the peripheral tissues. Examination of the insulin-like activity of the serum by the rat diaphragm method revealed no change in this activity after the administration of tolbutamide, and no difference was found in the rate of disappearance of intravenously administered radioiodine (I^{131}) from the plasma of normal rabbits after the intravenous injection of tolbutamide. These studies suggest that tolbutamide does not produce its hypoglycemic action by increasing the level of insulin in the tissues and so promoting the peripheral utilization of glucose.

Heat Stroke from a Turkish Bath.—Stoddart (*Brit. M. J.* 1:443, 1958) reports that a woman of 57 was admitted unconscious to hospital after having taken a Turkish bath for 40 minutes at a temperature of 100 F (37.8 C). She appeared to be asleep in the bath but could not be roused and was sent by ambulance to hospital about two hours after entering the bath. Her rectal temperature was 107 F. (41.7 C), pulse rate 90, and blood pressure 90/50 mm. Hg. Her pupils were widely dilated, she did not respond to painful stimuli, reflexes were generally depressed, the plantar response was flexor, and there was marked opisthotonos. A diagnosis of heat stroke was made. She was given 25 mg. of chlorpromazine intramuscularly to depress the heat-regulating center and discourage shivering, and her temperature was further reduced by wet sheets and ice packs. There was no evidence of a neurological or cerebral vascular lesion. The patient developed generalized epileptiform convulsions, which were controlled by intramuscular injections of phenobarbital. Consciousness returned after seven hours, and she was discharged from the hospital on the fifth day. A follow-up showed no mental or neurological impairment.

Estimation of Blood Loss in Melena.—When erythrocytes labeled with radioactive chromium are introduced into the stomach or duodenum more than 90% of the chromium can be recovered in the feces. Jones (*Brit. M. J.* 1:493, 1958) has quantitatively estimated blood loss in the stools by labeling the subject's red blood cells with radioactive chromium (Cr^{51}), reinjecting them into the circulation, and subsequently estimating the Cr^{51} content of the stools with a Geiger-Müller counter. Six anemic pa-

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tients known to be losing red blood cells in the stools were studied. The patient's own erythrocytes were labeled with about 100 μc of Cr^{51}, using Cr^{51} with a specific activity of 10 to 30 μc per mcg. of chromium. The labeled erythrocytes were then injected intravenously and the loss of Cr^{51} in the stools estimated. The rate of loss of Cr^{51} from the blood stream was increased beyond the normal figure, based on red blood cell survival estimates, and this was almost completely accounted for by the amount of Cr^{51} lost in the stools. The amount of Cr^{51} in the stools did not completely account for the rate of loss of Cr^{51} from the blood stream, indicating that some Cr^{51} is reabsorbed from the gastrointestinal tract but is not reutilized. Thus the Cr^{51} method slightly underestimates the amount of blood lost in the stools. In two control subjects known not to be bleeding into the gastrointestinal tract, the amount of Cr^{51} excreted in the stools following the intravenous injection of cells labeled with Cr^{51} was very small, being equivalent to 0.1 to 0.2 ml. of red blood cells daily. Because of this slight excretion the daily loss of blood in the stools must be more than 1 ml. before it can be detected by this tracer method.

Advanced Mammary Cancer.—Hypophysectomy causes improvement in 50 to 67% of patients with advanced mammary cancer, but there is usually a hypophyseal remnant, as complete extirpation can never be certain. Edelstyn and co-workers (*Lancet* 1:462, 1958) described a method for complete destruction of the pituitary by combining surgical removal with subsequent irradiation of the pituitary fossa with yttrium-90 oxide. Hypophysectomy is accomplished by the right transfrontal route, and when as much of the gland as possible has been removed and hemostasis secured, the fossa is packed with 7.5 to 10.0 mc of radioactive yttrium (Y^{90}) oxide mixed with dental impression wax. A wax pellet is rolled between two thick flat pieces of Perspex into a cylindrical shape and loaded into a piece of glass tubing, 30 cm. long and 0.5 cm. in internal diameter, which is fitted with a metal plunger so that the wax can be placed in the pituitary fossa. Great care is needed to prevent radioactive contamination of operating room personnel. Contaminated instruments are stored until they become inactive. Cortisone is given to the patients starting two days before the operation and treatment with thyroid extract is started about a month later.

Twenty-five patients have been so treated. Five died postoperatively, and of the survivors 16 have shown objective evidence of remission. Complications include major damage to the optic nerve in two patients and third cranial nerve lesions in four, probably due to forcing of the wax through the lateral wall of the fossa. Thyroid function was depressed in all patients who were well enough to be examined, and only 1 patient in 19 showed evidence of estrogenic activity, as judged by vaginal cytology. Autopsy was performed on three of the five patients that died, and there was evidence in two of them of still viable pituitary tissue.

Single Massive Doses of Penicillin.—Joseph (*Brit. M. J.* 1:487, 1958) compared the results of using single massive doses of penicillin for the treatment of acute lower respiratory infection in children with those of continuous therapy. The diagnosis was based on radiologic appearances as well as on physical signs. A single dose of 0.2 to 0.5 Gm. of sodium penicillin was given intramuscularly to 52 children, most of whom received in addition 0.3 Gm. of sulfadimidine per kilogram of body weight. The 54 children in the control group were given sodium penicillin every 6 or 12 hours in the standard dosage and 0.15 to 0.2 Gm. of sulfadimidine per kilogram per 24 hours, administered every 6 hours in divided doses. Treatment was continued in this group for at least five days. Comparison of the effectiveness of the two forms of treatment was based on the successes, failures, relapses, and complications. In addition the effect of treatment was observed on the fall in temperature, pulse, and respiratory rates; the rate of disappearance of physical and radiologic signs; and the length of stay in hospital. On these criteria the results obtained in the children receiving a single massive dose of penicillin were as good as those in children under standard continuous penicillin treatment. In children particularly, 1 injection of penicillin compared with 20 or more is an advantage. Apart from pain and discomfort, all the hazards associated with injections given in the hospital are avoided, the time of the nursing staff is saved, and patients are not disturbed in their sleep for injections.

Extracellular Water in Epilepsy.—The experimental production of convulsions by overhydration and their control by dehydration is well known. A dietary regimen involving dehydration has also been used with some success in the control of epilepsy with improvement in the electroencephalogram. Olbrich and co-workers (*Brit. M. J.* 1:500, 1958) investigated the relation between the distribution of extracellular water and total body water in epileptic

and nonepileptic subjects. Eighteen epileptic males, with a duration of disease of 2.5 to 40 years, and 38 nonepileptic males were studied. The subjects were prepared for the test by being given a low-protein diet and ample fluid the day before. The total body water was measured by the urea method of McCance and Widdowson, but no significant difference was found in the values for the two groups. There was, however, a significant difference between the absolute values for extracellular water, measured by the sodium thiosulfate method, the amount in epileptics being 12.5 ± 3.1 liters, and in normal subjects 11.3 ± 1.76 liters. When the ratio of extracellular water to total body water expressed as a percentage was compared in the two groups the significance of the difference between them became more evident. This ratio was 2.9 ± 5.1 in the former and 25.3 ± 3.4 in the latter group. When the epileptics were classified according to frequency of the fits, those with the greatest frequency had the highest ratio of extracellular water to total body water. The average total sodium in the extracellular space was also greater in the epileptics.

As all the epileptics were investigated in the interval between attacks, the results suggest that an increase of extracellular fluid is always present in epileptics and that it is this that predisposes them to seizures. The beneficial effects reported of treatment by starvation, dehydration, ion exchange resins, and acetazolamide may depend on loss of fluid and sodium from the extracellular space. Restriction of sodium in epileptics would be rational treatment, according to these observations.

Army Medical Museum.—The Royal Army Medical Corps Historical Museum at Crookham houses a collection that is mainly for the nonspecialist. No effort has been made to duplicate the purely technical collections that already exist. This group of apparatus, models, plaques, letters, drawings, photographs, standards, and medals illustrates brilliantly the development of the army medical services from the time of the Peninsular War, in which the Duke of Wellington had small medical resources at his command, to the present. The collection reveals doctors, in war and in peace, as great collectors. The trophies range from a Russian drum won at Sebastopol to a Coptic cross collected by a surgeon in Lord Napier's Abyssinian Campaign of 1868.

Parking Problems.—The St. Marylebone Borough Council in London has asked the Minister of Transport and Civil Aviation to ban parking in an area of London in the vicinity of Oxford Street except at parking places where a charge by parking meters would be made. Four local physicians and the British Medical Association were among the objectors and, as reported in the *British Medical Journal* of Feb. 8, attention was drawn to the difficulties of doctors in parking and to the fact that in some cases the difficulty was so great that doctors had been forced to refuse to take some people as patients. It was pointed out how difficult it would make the life of a general practitioner who must have his car available at all times. It was suggested that exemption from payment of essential persons such as doctors and midwives on duty should be arranged.

Marriage and Divorce 1956.—The Registrar General's Statistical Review of England and Wales for the year 1956 reported that in 1956 there were 352,944 marriages, giving a rate of 15.8 persons marrying per 1,000 of population; 120 of the men and 2,681 of the women were under 17 at marriage, and 5 men, though no women, were over 90. The five men were all widowers. The commonest age for men to get married was 23 and for women 21. There is a tendency for people to marry younger than they did 50 years ago, and widows and widowers are much more likely to remarry than they were 50 years ago. Marriages dissolved by divorce in 1956 numbered 25,718 and by annulment 547. These figures are respectively about six times and three times the comparable figures for the 1930's. The figure for divorces was the lowest since the end of World War II. The commonest age for divorce to take place is 30 to 34 for both husbands and wives, and 10 to 14 years is the commonest duration of those marriages which end in divorce. Of those couples who were divorced, the largest number (8,934) had no children, and the next largest number (8,212) had only one child. In 188 cases of divorce there were seven or more children of the marriage.

Bodies for Dissection.—The minimum number of bodies required each year for dissection is 622. In the last two years more than this number has been supplied, but the distribution is variable. The medical schools seriously short of such bodies are those at Cambridge, Newcastle, and Glasgow. For the last two years the supply in London has exceeded the 10-year average of 220, while in the provinces and Scotland it has been below the 10-year averages of 328 and 112 respectively. Last year bequested bodies comprised 70% of the London supply, 44% of the provincial supply, but only 15% of the Scottish supply, and of the total number of bodies available 48% were bequests.

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CORRESPONDENCE

FRAUDULENT SECURITIES PRACTICES

To the Editor:—During the course of various investigations conducted by the United States Securities and Exchange Commission, it has been reported that a considerable number of the members of the medical profession have been victimized by high-pressure telephone salesmen who have induced doctors, as well as members of other professional groups, to purchase securities of no value or dubious value.

As a result of this nefarious conduct on the part of these salesmen, the New York State Attorney General and the United States Securities and Exchange Commission developed and published a 10-point guide to alert the public against fraudulent securities practices. Notwithstanding the dissemination of the information and advice contained in a public pronouncement, there have been a mounting number of complaints brought to our attention concerning the continued activities of unscrupulous stock salesmen.

The Securities and Exchange Commission News Digest, Nov. 26, 1956, covers this subject matter. We believe that it will be an effective guide for the members of the medical profession in avoiding the pitfalls involved in the purchase of securities from unknown sources.

New York State Attorney General Jacob K. Javitz and the U. S. Securities and Exchange Commission, jointly announced today that they have agreed upon a basic 10-Point Guide for the public, to furnish a warning against fraudulent securities practices.

Among those cooperating with the Attorney General and the Commission in developing this 10-Point Guide were representatives of the New York Stock Exchange, American Stock Exchange, National Association of Securities Dealers, Association of Stock Exchange Member Firms, Investment Bankers Association of America and the Better Business Bureau of New York City, which originally met on September 28, 1956. This meeting was called by Mr. Javitz and Mr. Paul Windels, Jr., New York Regional Administrator of the Securities and Exchange Commission.

Because of the mounting number of complaints being received by law enforcement agencies involving unscrupulous stock operators, it was felt that a combined effort supplementing the continuing activities of the cooperating organizations to warn the public of the pitfalls involved in buying securities from unknown sources must be made.

"Too many people have fallen prey to unknown, high-pressure telephone salesmen who have peddled securities of no value or dubious value," Mr. Javitz and Mr. Windels declared. "With the publication of this 10-Point Guide," they added, "we hope to provide the public with a simple but effective guide to saving themselves from falling for sucker bait."

The 10-Point Guide is as follows:

1. Think before buying.
2. Deal only with a securities firm which you know.
3. Be skeptical of securities offered on the telephone from any firm or salesman you do not know.
4. Guard against all high pressure sales.
5. Beware of promises of quick spectacular prices rises.
6. Be sure you understand the risk of loss as well as the prospect of gain.
7. Get the facts—do not buy on tips or rumors.
8. Request the person offering securities over the phone to mail you written information about the corporation, its operations, net profit, management, financial position and future prospects. Save all such information for future reference.
9. If you do not understand the written information, consult a person who does.
10. Give at least as much thought when purchasing securities as you would when acquiring any valuable property.

This 10-Point Guide has been one of the developments of the close cooperation initiated between the offices of Attorney General Javitz of New York and the New York Regional Office of the Securities and Exchange Commission headed by Paul Windels, Jr.

It is presently contemplated that thousands of copies of the above 10-Point Guide will be distributed throughout New York by securities firms, the Better Business Bureaus, and other agencies interested in increased vigilance on the part of the investing public.

PAUL WINDELS JR.
EDWARD SCHOEN JR.
United States Securities and Exchange Commission
225 Broadway, New York 7.

AID TO FOREIGN MEDICAL LIBRARIES

To the Editor:—Medical libraries and medical schools abroad are rapidly organizing and growing in Asia, Latin America, and other parts of the world. The United States government and private American aid have been helping through technical aid and scholarships to encourage and foster this growth. American medical practices and knowledge are thus of aid all over the world in making a better life possible. Many members of the American Medical Association appear to have been concerned directly or indirectly with this program of development. Those who have traveled abroad and visited medical libraries and institutions know that one of the most constant needs, always present in spite of all that has already been accomplished, is for up-to-date medical publications. Textbooks, new studies in all branches of medicine and public health, and professional journals are much in demand and impossible to provide in the numbers needed.

The A. M. A. recently helped meet this worldwide need by donating several thousand surplus copies of THE JOURNAL and the Archives to the dis-

tribution program of the U. S. Book Exchange, Inc., a private library service organization located in Washington, D. C. Through contract funds provided by the International Cooperation Administration and the U. S. Information Agency, this organization will distribute the journals (all published within the last two years) to institutions abroad for use in their libraries and as textbooks in their classrooms. This gift will be of inestimable value to the institutions receiving the journals. This is an ever present need, and the U. S. Book Exchange can receive and distribute almost any number of professional journals of the specialist variety and any books or monographs containing information still useful in actual practice. At present, the agency has enough of the general journals to satisfy distribution needs. Cost of transportation by parcel post or freight to Washington and the value of the publications donated are deductible for income tax purposes. Inquiries or donations should be addressed to the U. S. Book Exchange, Inc., 1816 Half St., S. W., Washington, D. C.

Alice Dulany Ball
United States Book Exchange, Inc.
Library of Congress Building
Washington 25, D. C.

DIETS

To the Editor:—Many physicians have been confused by publications in the lay press (De Groot: *Look* **20**:36-39 [June 12] 1956; Norman: *Ladies Home Journal* **73**:44-45 [July] 1956) and by conflicting letters (Dole: *J. A. M. A.* **161**:901 [June 30] 1956; ibid. **161**:1700 [Aug. 25] 1956) that have appeared in the correspondence section of The Journal regarding two diets studied at the Rockefeller Institute. This letter is intended to clarify the situation. The first of these diets (the so-called Revolutionary Rockefeller Diet) used regular foods whose protein content was restricted on the assumption that a low-protein intake would reduce appetite. The scientific report (Dole et al.: *Am. J. Clin. Nutrition* **2**:381-391 [Nov.-Dec.] 1954) of this work supported but did not prove the assumption and indicated that the diet had been unsuccessful when the patients left the hospital. The second diet (the so-called Crash Diet or Fabulous Formula) replaced natural foods by a formula mixture whose operational principle was its fixed, invariant prescription of a low-caloric intake. The mixture was one of many (Ahrens et al.: *Am. J. Clin. Nutrition* **2**:336-342 [Sept.-Oct.] 1954) designed for hospital metabolic studies, and its constituents were chosen for convenience and taste. For use in weight reduction, the total intake was reduced to 900 calories a day. The relatively low protein content which resulted was a property of the original mixture and was not a deliberate choice. Formula mixtures of different composition have been used in this way with similar results. The formula diet has no common application or mode of action with the low-protein diet. The only relationship between the two diets is that they were studied at different times in the same institution.

A later paper (Joliffe: *J. A. M. A.* **161**:1633-1636 [Aug. 25] 1956) implied that both diets were unsafe because of their low protein content. I have had no direct experience with the low-protein diet. The formula diet studies have appeared in abstract form (Feinstein et al.: *Clin. Res. Proc.* **5**:17-18 [Jan.] 1957; Feinstein and Schwartz: ibid. **5**:180 [April] 1957) and a full scientific report (Feinstein et al.: *Am. J. Med.* **48**:330-343 [Feb.] 1958) has been published. The results show that patients dieting with formulas have maintained the same range of nitrogen balance as those on regular high-protein diets and have preserved normal values in all the physiological variables tested. In the outpatient stutlies, the percentages of weight loss in the formula-fed group were much higher than those obtained with many conventional diet programs.

The purpose of this letter is not to discuss methods or desirability of maintaining nitrogen balance during temporary periods of weight loss, nor is it to debate the plethora of nutritional theories and paucity of scientific data regarding techniques and results of weight reduction programs. It certainly does not intend to advocate the routine use of formula diets in obese patients. Its purpose is rather to call physicians' attention to factual results which verify the safety and utility of a formula diet in obese patients. Diets using conventional food are accepted and recommended by nutritional tradition. They are generally desirable for overweight patients but have often been unsuccessful. A formula diet has been strikingly effective in certain instances where regular diets have failed, and it provides an alternative therapeutic approach in some of these situations. Its possible application in individual patients should be determined by the physician without unwarranted fears for its safety.

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New York.

WEIGHT REDUCTION

To the Editor:—Dr. Max Wishnofsky (*J. A. M. A.* **166**:964-965 [Feb. 22] 1958) is in error in stating "The thesis of Pennington is that when an obese individual is on a high-fat, high-protein ad libitum diet, a substantial loss of weight will occur even

when there is no negative caloric balance." Such a statement appears nowhere in any of the papers I have written, nor is it implied in them. On the contrary, my statements on the matter have been very clear. For example: "In the treatment of obesity, the aim of which is to reduce the size of the adipose deposits, it is axiomatic that the caloric expenditure must exceed the intake" (*J. Clin. Nutrition* 1:100-106 [Jan.] 1953). Again: "The overall determinant in weight reduction is the degree to which the caloric expenditure exceeds the intake" (*New England J. Med.* 248:959-964 [June 4] 1953). My thesis has been summarized correctly by other commentators.

My thesis on weight reduction concerns itself with the manner in which a negative caloric balance is to be brought about. According to my plan, "treatment would be directed primarily toward mobilization of the adipose deposits, and the appetite would be allowed to regulate the intake of food needed to supplement the mobilized fat in fulfilling the energy needs of the body" (*J. Clin. Nutrition* 1:100-106 [Jan.] 1953). The evidence that such a plan of treatment, alternative to the usual one of caloric restriction, is possible has been presented in a number of my papers. I would like to mention the evidence of Kekwick and Pawan (*Lancet* 2:155-161 [July 28] 1956; *Metabolism* 6:447-460 [Sept.] 1957) that the metabolism of the obese person rises after a fatty meal.

Anyone who is interested in a correct statement of my thesis and in analyzing it for himself might refer to my papers, some of which are listed at the end of Dr. Thorpe's article (*J. A. M. A.* 165:1361-1365 [Nov. 16] 1957).

Alfred W. Pennington, M.D.
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DIAGNOSIS OF SKIN TUMORS

To the Editor:—May I add certain observations to the fine paper on "Aids in Early Diagnosis of Tumors on Tip of Nose" by Haggart and Rebello (*J. A. M. A.* 166:1010-1013 [March 1] 1958)?

In examining the skin changes of the eyelids and nose, one useful bit of equipment (which is available wherever an opthalmologist is found) is a biomicroscope or slit lamp. This instrument provides varying magnifications with a stereoscopic microscope of very high correction and a source of illumination, which can be used to examine the skin. I can recommend to all who have an ophthalmologist as a friend that skin lesions on the face be examined with his biomicroscope. As pointed out by the writers of this paper, skin changes are differentiated with powerful illumination.

My own personal findings with the additional use of Wood's light (long ultraviolet, 3,650 A) and stereobiomicroscopy have revealed further important changes which are of assistance in deciding (prior to any biopsy) whether or not a skin alteration is possibly neoplastic. The findings are embodied in the paper on "Ophthalmoluminescence: Definitions and Descriptions of Effects" (*Am. J. Ophth.* 44:360-372 [Sept.] 1957). It should be stressed that all lesions of the lid, nasal bridge, nasal tip, the facial area, the ears, and adjacent tissues are easily examined with high magnification with the biomicroscope, and with the use of Wood's light a further diagnostic aid is secured. It is to be hoped that many practitioners who do not employ optical magnification for skin observation will do so in the future, particularly in examining the facial tissues.

Lester Stein, M.D.
203 Sinclair Bldg.
Steubenville, Ohio.

DOCTORS AS CITIZENS

To the Editor:—This is to extend my heartiest congratulations on "Doctors and Politics," which appeared in The Journal, March 15, 1958, page 1313. The article was timely, well-written, and interesting. It was a powerful and worthy attempt directed against the "myopic lethargy" which, according to the author, seems to beset many physicians and, I think, also many medical students. The article says doctors "must take an active part in the political life of their communities and of the nation" if they wish to preserve their individual freedom. They "must emulate the tradition of the past," wherein physicians "were imbued with the idea of personal freedom, . . . were distinctly individualistic in their ideas and actions." Doctors "act as if centralized power, compulsion, and over-all planning (by the federal government) were the source of our economy. . . . 'The mainspring of human progress is individual liberty, and individual liberty decreases as federal power increases.'"

I particularly agree with these sentiments and believe that physicians, as acknowledged leaders of the community, should help spark their fellow-citizens' interest in reevaluating the "anything goes" philosophy which permeates our government and society at many levels. Recalling our history, we realize that national survival begins to be endangered as an inevitable consequence of easy-come, easy-go prosperity and a politically lackadaisical attitude of the people.

Earl DeGrey Short Jr.
The Medical College of South Carolina
Charleston, S. C.

MEDICAL FILM REVIEWS

RX—A Prescription for Better Hospital Procedures: 16 mm., color, sound, showing time 34 minutes. Prepared in 1957 by and procurable on loan or purchase from University of Illinois Research and Educational Hospital, 840 S. Wood St., Chicago 12.

The purpose of this film is to record the actual steps taken in developing a work simplification program at the University of Illinois Research and Educational Hospital. Several sequences describe the training and instructional sessions in which the students participate. These sessions include the pegboard demonstration, the session on the "open mind to change," the five-step pattern of work simplification, and the use of the flow-process chart. Activities of the problem-solving team are illustrated through two actual improvements in procedures in the hospital. These are presented in "before and after" fashion—the old versus the new method of carrying out these two procedures. The photography is satisfactory, although further editing would have improved the film. It should be of interest not only to hospital administration and personnel but also to medical staff members. The work simplification approach to more effectively provide patient care and the stimulus to rethink routine should be of assistance to all concerned with care of patients in hospitals.

Human Gastric Function: 16 mm., color, sound, showing time 19 minutes. Prepared by Stewart Wolf, M.D., Oklahoma City. Produced in 1957 by Sturgis Grant Productions, New York, and procurable on loan from Medical Film Center, Smith, Kline and French Laboratories, Philadelphia 1.

The purpose of this film is to teach and demonstrate the effects of different psychological states and stresses on gastric function. It is a report of an experimental study on "Tom," a unique patient widely known in medical circles. "Tom" had an accident in early childhood which resulted in an extensive gastric fistula. This condition allowed study of the mucosa and observation of secretory action and gastric motility under various conditions. For example, the investigators were able to gain insight into the stomach's complex responses to different psychological stresses. The film is a partial record of 15 years' continuous study of this patient. The effects of fear, anger, and serenity are dramatically shown. Observations on the protective action of gastric mucin and on the experimental production of a small, superficial peptic ulcer are included. This film is highly recommended for medical students and general practitioners. Gastroenterologists, internists, and psychosomaticists will also be interested.

NEW FILM ADDED TO A. M. A. MOTION PICTURE LIBRARY

The Other City: 16 mm., color, sound, showing time 22 minutes. Produced in 1957 by the American Cancer Society, New York. Procurable on loan (service charge $1.00) from Motion Picture Library, American Medical Association, 535 N. Dearborn St., Chicago 10.

Using the theme, "A statistic is hard to remember; a city is hard to forget" and giving dramatic emphasis to the fact that about 75,000 American lives are needlessly lost to cancer each year, this documentary film shows a city of this size which has been figuratively erased from the living. The number 75,000 represents those who could be saved by presently known techniques of surgery and radiation if they got to their doctors in time for early enough treatment. Actual residents of Racine, Wis., not actors but ordinary people, lend themselves to human everyday vignettes which dramatize cancer's danger signals. It is pointed out that "the ways of their lives were various, but the way of their death was the same; needless." By natural symbols, buds blooming, streams dividing, and life bursting, the how if not the unknown why of normal and abnormal growth is told. This is a well-produced and well-developed film which is highly dramatic in its efforts to induce persons to pay attention to the warning signs of cancer. It would be effective in inducing persons to see their physicians frequently for purposes of cancer detection, and it is recommended for showing to adult audiences, including high school and college students.

Esophageal Perforation in a Sword Swallower: 16 mm., color, sound, showing time 9 minutes. Prepared in 1957 by and procurable on loan from Philip Thorek, M.D., 850 W. Irving Park Rd., Chicago 13.

This is a case presentation of a traumatic perforation of the esophagus, inadvertently self-inflicted by an apprentice sword swallower while practicing his art. An esophagogram and an artist's drawing showing the esophagus with the contained sword in relationship to the surrounding viscera demonstrate the site and manner of the perforation. The operative procedure of repair is shown. Although clear definition of the anatomic structures is lacking in some sequences, this is aptly compensated for by the interpolation of excellently colored drawings. The film ends with a professional demonstration of sword swallowing, executed by a tattooed master of the art. Roentgenograms showing the sword in place within the lumina of the esophagus and stomach attest that the deed is indeed authentic and not a feat of visual deception. The film is of specific interest to the surgeon who deals with problems of the esophagus and of general interest to the physician, resident, and intern as well as to apprentice sword swallowers.

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ILLUSTRATED FILM REVIEWS

Illustrated reviews of current films will appear when possible as a regular feature in THE JOURNAL. *They will consist of an introduction by the author followed by a pictorial digest of actual frame enlargements selected as key points of the film.*

RALPH P. CREER, *Director*
Motion Pictures and Medical Television, Council on Scientific Assembly.

HUMAN GASTRIC FUNCTION

Stewart Wolf, M.D.

This film presents an unusual case, illustrating only a few of the studies and experimental methods which have been reported by the author and published in book form. Some of the more important observations on gastric function are mentioned and certain early hypotheses are corroborated and elaborated.

The patient, "Tom," in 1895, at the age of 9, developed a complete esophageal stricture from swallowing scalding clam chowder. A gastric fistula was created, through which he has fed himself ever since. In the past 15 years, Tom has been the subject of extensive investigations—he thus may be compared with Alexis St. Martin, whose fistula was the object of the well-known scientific observations of Colonel Beaumont more than a hundred years ago. The written review of this film appears on page 2216 of this issue.

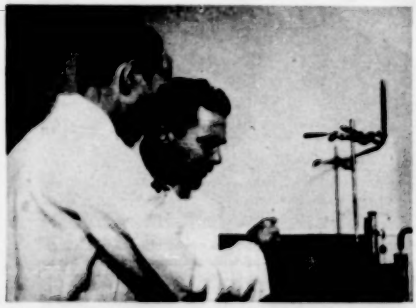

Fig. 1.—Study began at New York Hospital in 1943, in collaboration with Dr. Harold Wolff, and was continued by author at Oklahoma Medical Research Institute.

Fig. 2.—Tom became famous experimental subject. Over years his patience permitted extensive studies of his fistula, often involving observations continuously for days.

Fig. 3.—Fistula has permitted direct observation of gastric mucosa under many conditions and has allowed direct access to stomach for experimental work.

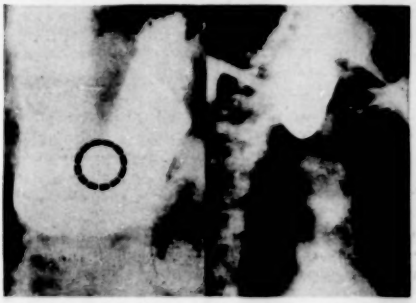

Fig. 4.—Left, gastric fistula is located about two-thirds of way between cardia and pylorus. Right, obstructed upper esophagus ends in cul-de-sac.

Fig. 5.—In order to eat, Tom chews food in ordinary fashion, then spits it into funnel connected to rubber tube which he inserts into his stomach.

Fig. 6.—When food was placed into stomach without being tasted, Tom lost weight, which implies that metabolic balance may be influenced by meaning of life experiences.

Fig. 7.—In studying appearance of exposed gastric mucosa, dissecting microscope and hand lens, as well as naked eye, were used.

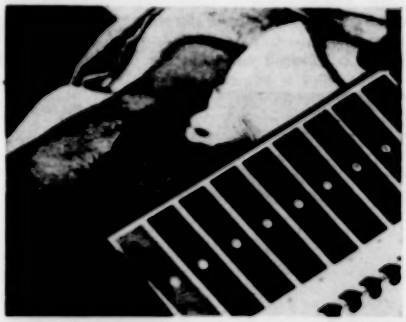

Fig. 8.—It was found that color could be evaluated accurately by comparing mucosa with ordinary Tallquist hemoglobin scale.

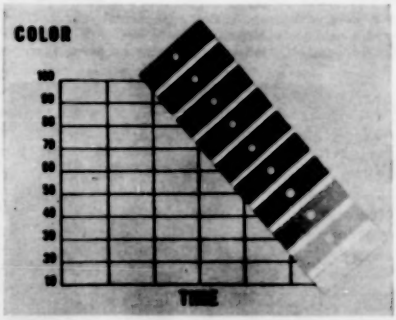

Fig. 9.—Thus, color of the mucosa, which obviously depends on blood flow, could be recorded at any time as number on graph.

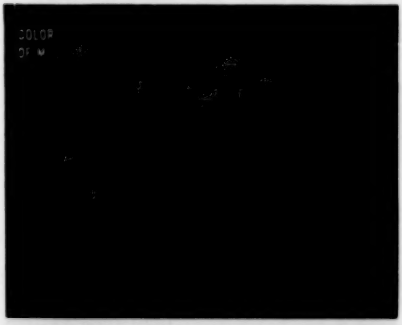

Fig. 10.—With use of special thermocouple, exact correspondence was found between amount of blood flowing through mucosa and its color.

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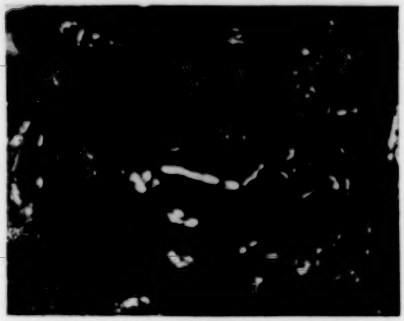

Fig. 11.—In pale, inactive state, large number of folds could be counted on surface of gastric mucosa.

Fig. 12.—When mucosa became hyperemic, neighboring folds merged, showing that with hyperemia goes turgor.

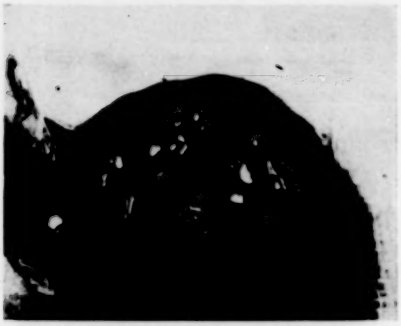

Fig. 13.—Turgid mucosa was vulnerable, slight touch causing petechial hemorrhages, and pain-sensitive, whereas it was insensitive in its pale state.

Fig. 14.—Gastric juice, easily collected for analysis with ordinary Chetwood syringe, quickly digests normal, healthy, tough skin.

Fig. 15.—However, thick layer of mucus on surface of stomach evidently protects delicate cells of mucosa against digestion by gastric juice.

Fig. 16.—Likewise, no damage could be observed, even under microscope, when strong acid or blistering mustard were tested on mucosa.

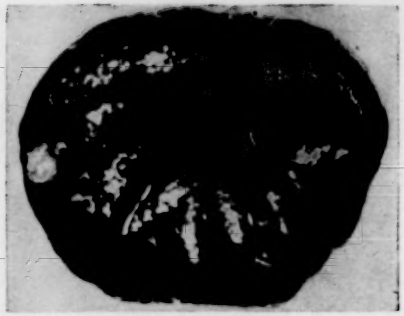

Fig. 17.—When mucus was constantly wiped from small area, crater developed, similar to clinical peptic ulcer. This healed rapidly when covered by petrolatum dressing.

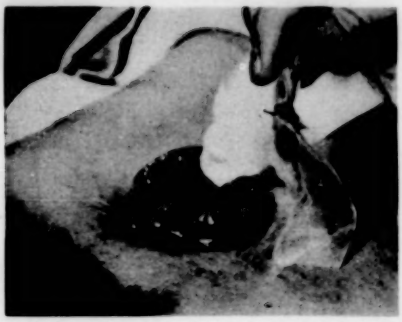

Fig. 18.—Another aspect of gastric function is motor activity, recorded by balloon inserted into Tom's stomach, other end being attached through tube to kymograph.

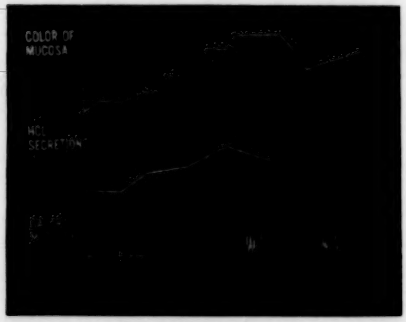

Fig. 19.—Records could thus be obtained correlating gastric motility simultaneously with inspection of color and chemical testing under variety of conditions.

Fig. 20.—Most interesting studies involved reaction of Tom's stomach to stress, occurring naturally or from interviews directed toward stressful response.

Fig. 21.—For example, when incident was recalled at which Tom became very angry, normal mucosa (above) became turgid and hyperemic (below), indicating hyperfunction.

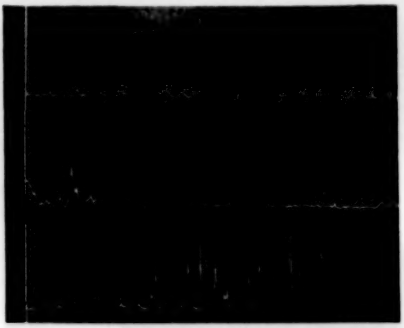

Fig. 22.—Tom has demonstrated that not only gastric hypofunction, as stated by Cannon, but potentially more dangerous hyperfunction may occur in reaction to emotional stress.

MEDICAL LITERATURE ABSTRACTS

INTERNAL MEDICINE

The Significance of Atrioventricular Block Complicating Acute Myocardial Infarction. D. B. Cohen, L. Doctor and A. Pick. Am. Heart. J. 55:215-219 (Feb.) 1958 [St. Louis].

The authors report on 43 men, of the average age of 58.4 years, and on 25 women, of the average age of 65 years, with acute myocardial infarction complicated by high degrees of atrioventricular block. Electrocardiograms revealed the posterior wall as the site of the infarction in 51 (75%) of the 68 patients, the anterior wall in 16 (24%), and both the anterior and the posterior wall in 1 patient. Thirty-two patients (47.1%) died within 2 months of the acute attack. Of these, 12 had an infarct of the anterior wall, 19 of the posterior wall, and 1 of both the anterior and the posterior wall. Thus, 75% of the 16 patients with infarction of the anterior wall died within a 2-month period, whereas 37.3% of the 51 patients with infarction of the posterior wall died within the same period. Thirty-three of the 68 patients had second-degree auriculoventricular block, 22 had advanced second-degree atrioventricular block, and 13 had complete block. Eleven (33%) of the 33 patients, 11 (50%) of the 22 patients, and 10 (77%) of the 13 patients died within 2 months after the acute attack. Autopsy reports were available on 17 patients who died within 2 months of infarction complicated by block. There was involvement of the interventricular septum by the acute infarct in 16 of the 17 patients. Twenty-nine (44%) of the 68 patients survived from 2 months to 1 year, 22 (34.9%) survived from 1 to 5 years, 7 (11.8%) survived from 5 to 10 years, and 1 survived more than 10 years. These data show that the immediate mortality rate for infarction with second-degree, advanced second-degree, and complete auriculoventricular block exceeds that for myocardial infarction in general, especially if the infarct is located in the anterior wall and the block is due to necrosis of the ventricular septum. The mortality rate increases with the severity of the block. The prognosis for myocardial infarction complicated by atrioventricular block is further aggravated by factors known to be of importance in determining the immediate mortality rate for infarction in general. The long-term prognosis in patients surviving the initial episode is not affected by the persistence of an atrioventricular block.

Family Studies of Respiratory Infections. F. S. W. Brimblecombe, R. Cruickshank, P. L. Masters and others. Brit. M. J. 1:119-128 (Jan. 18) 1958 [London].

The authors report results of a clinical and bacteriological study of respiratory illness, which was carried out in families of the same size and structure (2 parents and 3 children, 1 of whom was under 5 years of age), living in the same working-class area of London, in 3 types of dwelling, namely, self-contained houses, self-contained flats, and rooms in tenement dwellings. The study, which lasted 2 years, included regular visits by a pediatrician or a nurse who, besides making clinical and social observations, every 14 days took throat and pernasal swabs from all members of the family. Observations on the spread of "indicator" organisms within the family groups, such as pneumococci, streptococci, and staphylococci, were used to confirm or modify the epidemiologic indications about the introduction and spread of infection given by traditional clinical methods.

Of some 12 social and environmental factors examined, for example, maternal care, range of outside contacts, and dampness of the house, only overcrowding and, with less certainty, inadequate clothing were found to be related to the incidence of acute coryza and chronic catarrh. The increased clinical secondary attack rate in conditions of continued close contact observed in the more crowded families was confirmed by the higher rate of apparent transfer of type-specific pneumococci in these families. On clinical grounds alone, infections appeared to be most often introduced by the youngest or preschool child, that is, the most susceptible member of the family. Corrections for differences in susceptibility gave results which suggested that even in congested working-class districts the school child is the most frequently exposed to extrafamilial infection and the most vigorous spreader of infection within the family. The greater frequency of transfer of pneumococci and the higher carrier rates of hemolytic streptococci

The place of publication of the periodicals appears in brackets preceding each abstract.

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among schoolchildren supported this conclusion. Pneumococcic carrier rates were raised after the onset of acute coryza, but there was no evidence that the pneumococci were causally related.

Climatic changes appear to affect both the frequency of introduction of colds into households and the ease of transfer; the authors' findings and those of other workers suggest that this association is at least partly caused by changes in host susceptibility after abrupt seasonal changes in temperature and other climatic factors. Undue susceptibility to colds was shown to be a characteristic of individual children but not of families as a whole. Although some differentiation of syndromes which were presented as "acute coryza" and "sore throat" was possible on epidemiologic ground, sore throats often occurred without the presence of hemolytic streptococci in members of the family, and it seems likely that this syndrome results from more than 1 type of infection. A comparison of the experience of children who had undergone tonsillectomy with that of other children showed that, although streptococci were found less often in the throats of the children who had undergone tonsillectomy, the incidence of coryza and sore throat was identical in the 2 groups.

Treatment of Chronic Liver Disease with Crude and Deproteinized Liver Extracts. G. Barbagallo Sangiorgi. Minerva med. 48:3895-3908 (Nov. 21) 1957 (In Italian) [Turin, Italy].

Prolonged therapy with crude and deproteinized liver extracts diluted with fructose (levulose) was carried out intravenously, and in a few instances intramuscularly, in the case of 36 patients with liver disease. Fifteen of these patients had cirrhosis of the liver; 8, posthepatitis disturbances; 4, chronic hepatitis; and 9, liver stasis due to chronic decompensated circulation. The patients were tested for sensitivity, and all except 1, who was excluded from the series, tolerated the extract well. The dosage of the extract was gradually increased from 1 to 5 cc. a day within the first 5 days. The dosage of 5 cc. was then maintained until withdrawal of the therapy.

Among the patients with cirrhosis of the liver, improvement was observed in 8, no change in 3, and deterioration in 4. The improvement consisted of increase in diuresis, diminution of edema, and control of the ascitic fluid reaccumulation. This therapy afforded benefit to 3 patients with chronic hepatitis, but it was of no benefit to 1. The amelioration included retrogression of the icterus, increase in diuresis, and restoration of the functional capacity to normal. All patients with posthepatitis disturbances, who received this therapy, were clinically cured. Of the patients with liver stasis associated with chronic decompensated circulation, 6 had a rheumatic mitral endocarditic lesion and 3 had myocardosis. Medication with liver extracts brought about restoration of the blood circulation to normal in 7 patients, but there was no response in 2. However, this represented a remarkable therapeutic achievement in the case of the 7 benefited persons, because previous administration of the common cardiokinetic drugs to them had failed to ameliorate the cardiovascular status. The author points out that the most credible theory of the mechanism of liver extracts consists in their containing a growth-promoting factor, which stimulates the regeneration of the hepatic cells.

Clinical Manifestations of the Syndrome Characterized by Deficiency of Antibodies. S. Barandun, H. J. Huser and A. Hässig. Schweiz. med. Wchnschr. 88:78-82 (Jan. 25) 1958 (In German) [Basel, Switzerland].

The concept of the antibody deficiency syndrome includes diseases involving incapacity of the organism to form humoral immune antibodies. Persons who have this defect are extremely susceptible to bacterial infections, with the exception of tuberculosis, and they remain generally free from recurrences of virus diseases. On the basis of reports in the literature and of 20 cases observed by themselves, the authors differentiate between an isolated form and a combined form of the antibody deficiency syndrome. The isolated form is further subdivided into 3 groups: (*a*) congenital antibody deficiency with agammaglobulinemia which affects boys; (*b*) hypogammaglobulinemia with antibody deficiency in young persons and in adults of both sexes; and (*c*) normal gammaglobulinemic form of antibody deficiency in children and in adults. Of the 20 patients mentioned by the authors, 4 belonged to group a, 8 to group b, and 4 to group c. The 4 patients with the normal gammaglobulinemic form of the antibody deficiency syndrome (group c) included 2 boys and 2 men. Electrophoresis of the serum of these patients showed completely normal gammaglobulin values, while their susceptibility to infection was as severe as that of those who had agammaglobulinemia. The immunoelectrophoretic analysis showed lack of the beta 2A and beta 2M components.

In commenting on the combined forms of the antibody deficiency syndrome, the authors differentiate the form that concurs with various other diseases, particularly leukemias, myeloma, macroglobulinemia of Waldenström, diabetes, and nephrosis, from another form which hitherto has been observed only in the presence of gamma paraproteinemia and in which the gamma globulins not only are not lacking, or are not deficient, but may actually be present in excess, that is, hypergammaglobulinemia may exist. The authors observed this disorder in 2 of their 20 patients: in 1 man with

gamma plasmacytoma and in another man with atypical macroglobulinemia of Waldenström. Both of these patients showed severe hypergammaglobulinemia (paraproteinemia) and abnormal predisposition to bacterial infections, particularly bronchopneumonia, otitis, and infections of the upper air passages. The third form of the antibody deficiency syndrome is the transitory form, which is observed in early childhood, a time when hypogammaglobulinemia is a physiological condition. This transitory antibody deficiency syndrome that exceeds the physiological form is possibly caused by a retardation of the onset of the formation of gamma globulins and of antibodies, with increased susceptibility to infections. The authors observed this condition in 2 boys. Electrophoresis of the serum of these patients showed that both beta subfractions of the gammaglobulins were absent. The clinical and serologic symptoms of the antibody deficiency syndrome gradually regressed spontaneously in these boys.

Fifteen of the 20 patients were given mixed gamma globulins in relatively high doses, ranging from 0.5 to 1 ml. per kilogram of body weight at intervals of 3 to 4 weeks. The majority of the patients responded favorably to this treatment, irrespective of the serum gamma globulin content. The fact that the antibody deficiency syndrome may appear in patients with a normal serum gamma globulin content shows that deficient formation of antibodies is not necessarily associated with a reduced gamma globulin content. Electrophoretic analysis reveals that in most patients with the antibody deficiency syndrome the precipitation lines for beta 2A and for beta 2M are missing regardless of the behavior of the gamma globulins.

The Histopathology of the Antibody Deficiency Syndrome. H. Cottier. Schweiz. med. Wchnschr. 88:82-86 (Jan. 25) 1958 (In German) [Basel, Switzerland].

The immature developmental stages of the plasma cells, and to a certain extent also the lymphoblast cells of the mature lymph follicles, are generally regarded as the site of antibody formation. It was decided to study this substrate, particularly the lymphatic tissues, for tissue and cellular defects in patients with the antibody deficiency syndrome. This paper deals with histopathological studies (biopsy and, in 1 case, autopsy material) in 13 patients with the isolated form of the antibody deficiency syndrome. As a rule, the number of plasma cells in stimulated and nonstimulated lymph nodes and in chronically inflamed tissue paralleled the level of circulating gamma globulins. Some normogammaglobulinemic patients, however, showed an increased number of plasmocytes in lymph nodes, although antibody production was impaired. Structural defects of the lymphoreticular tissue (such as disturbed formation of the follicles, absence of secondary follicles, substitution of the lymphatic tissues by an increased number of reticular cells and by connective tissue, fibrous thickening of the capsule) were found in patients with agammaglobulinemia and hypogammaglobulinemia.

The extent of these alterations varied from patient to patient. Thus, there is no strict correlation between histological findings and the amount of circulating gamma globulins. In 1 patient who had the hypogammaglobulinemic type of antibody deficiency, the spleen, the lymph nodes, and the liver contained irregularly shaped accumulations and bands of epithelioid cells. Thus, the formation of epithelioid cells does not appear to be bound to the presence of humoral antibodies. Factors are cited which justify the assumption that the essential lymphocytophthisis (lymphocytic wasting) of Glanzmann-Riniker is identical with the defect in the autopsied case of agammaglobulinemia. It may represent a special form without sex-linked recessive heredity.

Colorado Tick Fever. R. H. Fitz and G. Meiklejohn. Postgrad. Med. 23:113-118 (Feb.) 1958 [Minneapolis].

The authors report on 2 patients, 1 a 38-year-old man and the other a 10-year-old boy, with Colorado tick fever. The case of the adult patient illustrates the prototypal form of the disease, with rapid onset of chilliness, retro-orbital headache, photophobia, general muscle aching, malaise, and a "metallic taste" in the mouth. The tip of the spleen was palpable 2 cm. below the left costal margin. Four days earlier the patient had been on a fishing trip in the Colorado Rocky Mountains. He had noted ticks on his body but had not found any attached. His symptoms progressed, and excessive weakness developed. His temperature ranged from 101 to 103 F (38.3 to 39.4 C) for 2 days. The 4th day he was afebrile, but symptoms returned the next day and persisted for 2 more days. Convalescence was somewhat prolonged and marked by excessive weakness and mild apathy for several days. The virus of Colorado tick fever was present in blood drawn on the 2nd, 3rd, 5th, and 6th days of illness. Neutralizing antibody against Colorado tick fever was present on the 16th day after onset. Complement-fixing antibody had not developed at that time.

The illness in the boy showed a more complex aspect, less easily recognizable on clinical grounds. It began with sudden malaise, fever, sore eyes, and headache which persisted for 2 days, and the patient's temperature ranged between 102 and 103 F (38.9 and 39.4 C). He was treated by the family physician with antibiotics and aspirin. He felt quite well on the 4th day and went to school but came home with a temperature of 103 F (39.4 C). On his

admission to the hospital he had a markedly stiff neck, and the tip of the spleen was palpable. The spinal fluid contained 90 cells per cubic millimeter (84% lymphocytes), 96 mg. of glucose per 100 cc., and 60 mg. of protein per 100 cc. The white blood cell count was 3,800 per cubic millimeter. The patient was irritable with slight injection of the right tympanic membrane and considerable injection of the scleras and conjunctivas. He had been in the mountains 1 week before the onset of illness. Two days before the illness his mother noted a tick embedded in the skin between his shoulder blades. Spinal fluid examination on the 3rd hospital day revealed 66 cells (all lymphocytes) per cubic millimeter and normal levels for glucose and protein; he was afebrile and was discharged. The virus of Colorado tick fever was grown from blood drawn on the 6th day of illness. Complement-fixing antibody was not present in the acute-phase serum initially but was present in a 1 to 16 dilution 6 weeks after the onset of illness. Without the history of exposure to ticks, this patient would have been considered to have nonspecific viral meningitis.

Colorado tick fever has a specific regional and seasonal incidence and a characteristic clinical pattern. The disease is caused by a virus transmitted to man by the adult hard-shelled wood tick, Dermacentor andersoni. The incubation period is usually 4 or 5 days. The illness occurs throughout the Rocky Mountain region from early spring to early autumn, with the peak incidence from April through the middle of July. With the increasing number of tourists and sportsmen visiting the Rocky Mountains, the incidence of the disease may well increase. At present the illness itself does not warrant extensive and specific measures to control the tick population, but this might be accomplished in endemic areas. Specific immunization is potentially available. Persons planning to enter areas which are heavily infested with ticks should be informed of the hazards and of appropriate personal precautions, including the wearing of proper clothing and careful inspection of scalp, neck, sleeve, belt, trouser, and sock-line areas for ticks at frequent intervals. Although the disease is contracted only in the Rocky Mountain area, physicians throughout the country should be aware of the clinical entity.

Chronic Unspecific Pneumonia: Clinical Picture. T. W. Aas and J. Krohn. Nord. med. 58:1949-1952 (Dec. 19) 1957 (In Norwegian) [Stockholm].

In spite of the application of the usual diagnostic methods, lung infiltrations of unknown etiology are not infrequently encountered. When they are limited to a localized part of the lung, the question is whether tuberculosis or a malignant process is present, possibly combined with atelectasis or unspecific pneumonia. In the course of a year 7 patients, 6 men and 1 woman, were treated for chronic unspecific pneumonia. One man was 30 years old; the other patients were over 45. The disease was localized in the upper part of the lung. In 5 cases the disorder set in with acute catarrhal symptoms, partly with the picture of pneumonia. Administration of the usual antibiotics did not give wholly satisfactory effect in any case. The later course was marked by cough, in some cases with hemoptysis, with periodic fever and, in part, greatly increased sedimentation reaction. Tests for tubercle bacilli were negative in all cases. The bronchoscopic picture was, on the whole, normal. Why a pneumonic process takes this course is not clear. In 3 patients resection of the affected part of the lung was done, as the possibility of cancer could not be completely excluded. Chronic pneumonia-like infiltration was found in the excised parts of the lung. Patients in whom cancer is suspected must be referred for operative treatment.

SURGERY

Scalene Node Biopsy: Implications in Abdominal and Thoracic Disease. F. A. Pualwan, C. D. Sherman Jr., G. L. Emerson and others. Cancer 11:4-8 (Jan.-Feb.) 1958 [Philadelphia].

Although the authors had performed scalene node biopsies before 1956, they then began to use a standard technique, which is carried out with the patient under local anesthesia through a 5-to-7-cm. incision placed about 1 to 2 cm. superior and parallel to the clavicle and centered over the lateral edge of the sternocleidomastoid muscle. The prescalene fat is exposed by retracting the sternocleidomastoid muscle medially and the omohyoid muscle laterally. When the dissection is extended medially to the junction of the internal jugular and subclavian veins, one easily obtains a chain of lymph nodes in the superior mediastinum. The authors obtained as many as 31 nodes in a specimen measuring not more than 4 or 5 cm. in diameter without there having been any clinically palpable nodes on physical examination. This report is limited to the first 100 patients for whom 102 scalene node biopsies were done from January, 1956, to January, 1957. Its primary aim is to broaden the indications for scalene node biopsies to include those cases in which neoplasm occurring outside the chest, particularly in the abdomen, is suspected and also to include those systemic diseases that may manifest themselves in the lymph nodes.

Twenty-seven of the 102 biopsies were carried out in the presence of abdominal lesions. This group included 20 patients with proved and probable cancer. None of these had any radiologic evidence of the disease in the lungs or the mediastinum. There were 4 positive scalene node biopsies; two of the specimens were from patients with carci-

noma of the stomach and 2 from patients with carcinoma of the pancreas. In the 74 biopsies done for intrathoracic disease, there were 34 patients with proved cancer, with 15 positive biopsies. The biopsy was negative for 11 of 12 other patients in whom malignant tumor seemed probable. The biopsy for the remaining 28 patients in the intrathoracic group proved benign for 24 and probably benign for 4. Only 4 of the 25 patients with positive biopsies in the entire group had clinically suggestive scalene nodes, and 2 of these had lymphoma with slight generalized lymphadenopathy.

There was only 1 patient in the entire group who had a scalene node biopsy for a generalized disease without gross evidence of disease either in the thorax or in the abdomen. This was a patient with generalized lymphadenopathy, for whom the diagnosis of giant follicular lymphoma was made on scalene node biopsy. A scalene node biopsy is of definite value when it is positive. A negative biopsy, however, does not rule out malignancy. The procedure permits a diagnosis in poor risk patients with probably inoperable abdominal or thoracic disease. A positive scalene node biopsy may change the surgeon's attack from a massive "curative" resection to a lesser "palliative" operation. Only 1 major complication occurred in the patients for whom the 102 biopsies were done; a chylous fistula, followed by a wound infection, developed after 1 specimen had been taken for scalene node biopsy. The resulting chylothorax required repeated thoracenteses but was controlled in about 15 days. The risk of a rare complication is regarded as justifiable in view of the great value of scalene node biopsy. Routine study by scalene node biopsy in patients with seemingly operable tumors may help to elucidate the natural history of cancer.

Internal Mammary Artery Ligation for Coronary Insufficiency: An Evaluation. R. Adams. New England J. Med. 258:113-115 (Jan. 16) 1958 [Boston].

The author presents a summary of observations made in an attempt to evaluate internal mammary artery ligation as a method of therapy for coronary insufficiency. Angina pectoris, myocardial ischemia and infarction, and major arrhythmias are regarded in this study as variable sequelae to inadequate coronary artery inflow. The histories of 4 patients are discussed. Three of the 4 patients believed themselves to be significantly benefited or relieved of pain by the operation. One patient had no pain. Pain disappeared in 2 cases before the vessels had been ligated but after the patients thought that they had been ligated. Pain relief in the 4th patient was related to lessened physical activity. Symptomatic improvement was not supported by objective clinical tests in any of the cases. One patient is dead, and the other 3 are believed to show changes indicative of progressive myocardial damage. Anatomic dissections were made in 3 fresh cadavers. The pericardiacophrenic vessels in these 3 subjects were variable. Mechanical measurements of intra-aortic and intramammary pressures and of internal mammary artery and pericardiacophrenic artery flow rates were made in 2 fresh cadavers. There was no measurable sustained increase in minute volume of flow from the pericardiacophrenic artery after occlusion of the internal mammary artery. The symptom-relieving effectiveness of a patient's will to succeed, of belief in his physician's word, and of confidence in his surgeon's procedure was again recognized.

Coarctation of the Aorta Proximal to Both Subclavian Arteries: Case Report of a Six-Year-Old Girl. I. Brynolf, C. Crafoord and E. Mannheimer. J. Thoracic Surg. 35:123-130 (Jan.) 1958 [St. Louis].

The authors report on a 6-year-old girl with a rare variant of coarctation of the aorta, proximal to both subclavian arteries, associated with a right-sided aortic arch and a left-sided descending aorta. The diagnosis was based on the absence of pulsations in the arms and legs and on the high carotid artery pressure. It was established preoperatively with the aid of selective angiocardiography. At surgical intervention, the coarctation was resected, and the aorta, which ran behind the esophagus, was transposed to run in front of the trachea. The patient withstood the operation well. A follow-up examination 5 months after the operation revealed that the carotid pulses were softer; the pulses in the left arm and both legs were palpable but faint. Pressure measurement in the carotid artery showed 110 mm. Hg as compared with 140 to 150 mm. Hg preoperatively. Angiocardiography revealed a fairly narrow communication between the ascending and the descending aorta and no postoperative aneurysm.

The patient was operated on at the age of 6 years, in contrast to the view of many workers that operation should be performed at the age of 10 to 20 years when sufficient collaterals have developed and before arteriosclerotic changes have appeared in the aorta. The recent tendency is to undertake operation on this malformation at an early age. At the authors' hospital in Stockholm it is done any time after the patient has reached 3 years of age. When signs of enlargement of the left side of the heart are present, it is performed even earlier, as soon as the anatomic details have been determined. Inasmuch as the subject of this report also had a cleft palate, both the vascular anomalies and the malformation of the palate fixed as the time at which fetal injury occurred the latter part of the second embryonic month. The patient had both a left-sided ductus arteriosus and a left-sided subclavian artery developed from the persisting thoracic aortic root on the left, and these may have

been the causes of her anomalies, according to Bedford and Parkinson's theory, but the existence of some other cause has not been ruled out.

Patent Ductus Arteriosus in Infants and Small Children. I. Boesen. Ugesk. læger **119**:1607-1609 (Dec. 12) 1957 (In Danish) [Copenhagen].

Patent ductus arteriosus was the main diagnosis in 17 out of 163 children under 2 years of age with congenital heart disease; 11 of the 17 were infants. The continuous murmur characteristic of patent ductus arteriosus is rarely heard in infants; yet it was present in 3 patients in this series. Heart catheterization (and possibly angiography) must therefore, as a rule, be resorted to for a definite diagnosis. Since from 10 to 15% of the cases of heart insufficiency, with fatal outcome in infancy, are due to patent ductus arteriosus and since the condition can be completely cured by operation, all children in this age group with heart insufficiency should be admitted for special examination. Of the 17 patients, 1 died of heart insufficiency. Sixteen patients were catheterized, and in 15 the catheter passed through the patent ductus into the aorta. Operative closure of the patent duct was done in the 16 patients, with 1 death.

Central Fractures of the Acetabulum. R. A. Knight and H. Smith. J. Bone & Joint Surg. **40 A**:1-16 (Jan.) 1958 [Boston].

The increasing frequency with which automobile accidents produce central acetabular fractures has brought a change in the attitude of orthopedic surgeons toward the treatment of these fractures. Resignation and defeatism have changed to active attack. Prior to 1949 at the Campbell Clinic in Memphis, Tenn., open surgery in central fractures of the acetabulum was limited to extracting the incarcerated head of the femur from the pelvis when manipulation and traction had failed to do so. The experience of these authors in the open reduction and internal fixation of these fractures began with a severely injured 19-year-old man. This patient had multiple fractures of the pelvis and ipsilateral fractures of the trochanteric region, shaft, and condyles of the femur, which precluded the use of the usual methods of traction. The acetabular deformity was so severe that joint junction would be poor, and degenerative changes would inevitably develop unless the acetabulum was restored. Accordingly, 16 days after injury the fracture was exposed through a Smith-Petersen iliofemoral approach. The operation, including exposure of the fracture, was difficult; it was almost impossible to close the fracture. Finally, Knowles pins were introduced into each fragment, one pin on either side of the fracture. These 2 pins were then grasped by a bone-holding forceps, and the acetabular fragments were forced into near-anatomic position. The reduction was maintained by 3 Knowles pins inserted across the fracture. All the patient's fractures united, and now, 8 years later, he has made a complete recovery with no hip disability.

Despite the satisfactory result in this patient, no further open reductions of central fractures of the acetabulum were performed at the Campbell Clinic until 1953. Since that time the authors have used this form of treatment in 7 more patients. The results have continued to be encouraging, and the authors are certain that this is the best method of treatment in properly selected cases. They comment on the mechanism of injury, anatomy of the fracture, roentgenographic studies, indications for operation, principles of reconstruction, and results. The primary objective is to restore the weight-bearing vault. Improvement of the symmetry of the pelvis or reduction of the posterior portion of the acetabulum, if achieved as well, is a dividend. Detailed classification of these fractures is neither possible nor necessary. A simple working rule divides them into 2 major types: horizontal and vertical. The horizontal type is approached through an anterior incision; the vertical, through a posterolateral incision. Stereoscopic anteroposterior roentgenograms are essential to the correct interpretation of the fracture and to the correct choice of approach. After open reduction after-care is greatly simplified, and morbidity is greatly diminished. The results after open reduction are, to date, generally superior to those obtained in the past by the older methods of manipulation and traction. Long-term studies of a large series of patients will be necessary before the method can be fully evaluated.

Indications for Common-Duct Exploration: Evaluation in 1000 Cases. M. K. Bartlett and W. R. Waddell. New England J. Med. **258**:164-167 (Jan. 23) 1958 [Boston].

The records of 1,000 unselected cases of cholecystectomy and choledochostomy at the Massachusetts General Hospital in the years 1943-1954 were reviewed, and the indications for and the results of the latter procedure were tabulated. The authors have attempted to determine the predictability of the presence of common-duct stones on the basis of the following indications: jaundice; stone palpable in the duct; dilated common duct; dilated cystic duct; small stones in the gallbladder; and previous attacks of pancreatitis. Previous attacks of pancreatitis constitute an indication for choledochostomy even though few common-duct stones will be found. Jaundice, "palpable stone," and dilatation of the duct system are sufficiently accurate indexes of stones in the common duct to be considered urgent indications for choledochostomy. The presence of small stones in the gallbladder, when this is the only reason for choledochostomy, constitutes a relatively poor indication; the opera-

tion has produced only a 16% yield of common-duct stones. It is in this group of cases that reduction in the frequency of choledochostomy is indicated.

Results of Electrosurgical Radical Mastectomy, with Routine Skin Graft and Postoperative X-ray Therapy: A Review of 309 Cases. D. Jackson and D. Jackson Jr. Cancer 11:18-21 (Jan.-Feb.) 1958 [Philadelphia].

End-result studies are presented for 309 patients with breast cancer treated by electrosurgical radical mastectomy, augmented by routine skin grafting and postoperative x-ray therapy. Emphasis is placed on the practice of extra-wide resection of skin requiring a routine skin graft. Use of the electrosurgical unit for both dissection and hemostasis is discussed, and the main value of this procedure as a time-saving feature is pointed out. Of the 309 patients operated on, 221 (71.5%) survived free of disease for 5 or more years; 85 (54.8%) of the 155 patients operated on 10 or more years ago have survived for at least 10 years. In a much larger series (913 patients) reported by Hickey, there was a 5-year survival rate of 46.8%. The authors feel that the more than satisfactory salvage rate achieved should give rise to careful consideration of the method of treatment used.

Surgical Treatment of Patent Ductus Arteriosus in Infants and Young Children. A. Gammelgaard and P. Therkelsen. Ugesk. læger 119:1609-1611 (Dec. 12) 1957 (In Danish) [Copenhagen].

Surgical closure of patent ductus arteriosus was done in 16 children, whose ages ranged up to 2 years; 9 were less than 1 year old. Pulmonary hypertension was most marked in the age group under 6 months. The patent ducts were completely closed by ligature and suture ligature. One death from respiratory insufficiency occurred 9 hours after the operation. In 5 cases there were transitory postoperative complications in the form of respiratory difficulties and cyanosis. Follow-up of 14 children showed no sign of recanalization. Measurement of pulmonary artery pressure during operation showed a fall in pressure but not, however, to normal values. Regression of pulmonary hypertension presumably depends on early closure. The operative mortality was low, considering the high incidence of pulmonary hypertension.

Sympathectomy for Raynaud's Phenomenon: Follow-up Study of 70 Women with Raynaud's Disease and 54 Women with Secondary Raynaud's Phenomenon. R. W. Gifford, E. A. Hines Jr. and W. McK. Craig. Circulation 17:5-13 (Jan.) 1958 [New York].

The authors report on 70 women, between the ages of 16 and 55 years, with Raynaud's disease. Sixty-eight of these patients were subjected to a sympathectomy for disease affecting the upper extremities, 16 of them being operated on for disease affecting both the upper and the lower extremities; the remaining 2 patients were operated on for disease affecting the lower extremities only. The period of postoperative follow-up for the 70 women varied from 1 to 28 years, and the mean follow-up period was 12 years. Sympathectomy for disease affecting the upper extremities gave good or excellent results in 37 (54%) of the 68 women. Good or excellent results were obtained more frequently if complications of Raynaud's disease, such as trophic lesions or sclerodactylia or both, were not present before the operation. There was no significant difference between the results obtained by preganglionic and postganglionic sympathectomies. Two patients (3%) lost portions of fingers after sympathectomy. Most of the 31 patients who had a fair or a poor long-term result of sympathectomy for disease affecting the upper extremities initially obtained a good result and then had relapses in the course of the first 2 years after sympathectomy. Sympathectomy for disease affecting the lower extremities gave good or excellent results in 17 (94%) of the 18 women.

In addition to the 70 women with Raynaud's disease, 54 women with Raynaud's phenomenon secondary to other diseases, such as acrosclerosis, rheumatoid arthritis, livedo reticularis, or acrocyanosis, were also subjected to sympathectomy on the upper or the lower extremities or both. The operation gave poor results in 33 (72%) of the 46 patients with secondary Raynaud's phenomenon in the upper extremities and only slightly better results in those in whom the lower extremities were affected. Sympathectomy should be reserved for patients with the more severe and progressive Raynaud's disease, since the prognosis is good without sympathectomy when the disease is mild or moderately severe and is not progressing.

Radiation Sterilization of Homogenous-Bone Transplants Utilizing Radioactive Cobalt: Preliminary Report. P. H. DeVries, C. E. Badgley and J. T. Hartman. J. Bone & Joint Surg. 40 A:187-203 (Jan.) 1958 [Boston].

Orthopedic procedures upon children and patients who are poor surgical risks demand that adequate supplies of sterile bone be available at all times. To assure sterility, bone transplants have been boiled or soaked in various types of antiseptic solutions. To assure adequate preservation of the tissue, they have been subjected to freezing and freeze-drying. Obtaining sterile bone at autopsy and subjecting it to freeze-drying have been an excellent method of maintaining a bone-bank. However, the technical difficulties involved in obtaining sterile bone are numerous, and the method does not always assure the surgeon that the tissue obtained is sterile. Freeze-drying does not sterilize

tissue and, in fact, has proved an excellent method of preserving many types of bacterial and viral organisms. The bone-bank problem is, therefore, how best to provide sterile tissue that is readily revascularized, absorbed, and replaced by the host with a minimum of reaction. Any process of sterilization or preservation that denatures the protein constituents of bone would probably retard the process of revascularization and absorption. It is known that high-intensity gamma radiation from radioactive cobalt will inactivate certain bacteria and viruses and that it is useful in the preservation of meat and other food products. Certain enzymes are only slightly altered by relatively large doses of gamma radiation.

Radiation sterilization of bone-bank bone was investigated by the authors, because the use of frozen bone and boiled or autoclaved bone had resulted in too high an incidence of postoperative wound infections and unsatisfactory results. Since December, 1954, radioactive cobalt has been used at University Hospital in Ann Arbor, Mich., to sterilize bone, blood vessels, and cartilage transplants. When combined with a freeze-drying process, radiation sterilization utilizing radioactive cobalt has proved an effective method of maintaining a bone-bank. It has been found that irradiated bone transplants function satisfactorily, both experimentally and clinically. In 104 surgical procedures in which irradiated homogenous-bone transplants were used, five postoperative wound infections occurred. The irradiated bone transplants were not felt to be the source of these infections. The encouraging over-all clinical results in 104 operative procedures prompt continued use of irradiated homogenous-bone transplants.

GYNECOLOGY & OBSTETRICS

Gestation and Carcinoma of the Uterine Cervix. H. Zeitz. Deutsche med. Wchnschr. 83:64-68 (Jan. 10) 1958 (In German) [Stuttgart, Germany].

The author believes that there is a causal connection between gestation and cervical carcinoma. He arrives at this conclusion from studies on 3,050 women with carcinoma of the uterine cervix in comparison with about 1 million women of the same age range in the general population. Information was obtained on (1) age of the women at time of marriage or first cohabitation and their number of births, (2) average number of births and age at time of marriage, (3) percentile distribution of births and age at time of marriage, (4) percentile distribution of births among Jewesses compared with other women, (5) incidence of cervical carcinoma and the occupation of the husband. No significant differences were observed between the women with and the women without cervical carcinoma as regards age at time of marriage or first coitus, but the women with cervical carcinoma had a much greater incidence of births. This greater incidence of births was evident in the women with cervical carcinoma regardless of age at time of marriage.

With regard to the occupation of the husband, it was found that the wives of laborers and farmers not only had a higher incidence of cervical cancer but also a higher average number of births than had the wives of office workers, professional men, and others at the higher social levels. On the other hand, even within the different social groups women with cervical carcinoma had a higher average number of births than did the other women. Among Jewesses, who in other investigations had generally shown a lower incidence of cervical carcinoma (owing to circumcision of the husband), cervical carcinoma was found more often in the high multipares than in the controls. The author deduces that gestation involves a factor that has some relationship to the genesis of cervical carcinoma. It is not the only factor, however. What processes in the course of gestation favor the carcinomatous degeneration of the uterine cervix await proof.

Adrenal Changes in Post-Term Infants and the Placental Dysfunction Syndrome. R. P. Bolande. Am. J. Path. 34:137-147 (Jan.-Feb.) 1958 [Ann Arbor, Mich.].

Physiological studies demonstrating a diminution in placental function near and after term have tended to corroborate the opinion of most investigators that prolonged gestation can be associated with deleterious effects on the fetus. The placenta is known to stop growing before term, and its permeability progressively diminishes after the 36th week of gestation. The autopsy records of the Institute of Pathology of Western Reserve University from 1952 to date were examined. Stillborn infants dying within 36 hours of delivery and newborn infants dying before 3 days of life, with the clinical diagnosis of prolonged gestation, postmaturity, or the placental dysfunction syndrome, were selected for study. Infants showing maceration or tissue autolysis were excluded. Criteria for postmaturity were based solely on calculated age of gestation and included infants of 41 weeks' gestation or more. The clinical diagnosis of the placental dysfunction syndrome was based on Clifford's criteria: evidence of recent weight loss; small size for the calculated length of gestation; a dry, wrinkled, scaling skin devoid of vernix caseosa; and presence of thick, yellowish meconium in the amniotic fluid, resulting in staining of the skin and of the umbilical cord.

Twelve newborn and stillborn infants with the placental dysfunction syndrome or signs of postmaturity were studied pathologically, particular attention being paid to the adrenals. In this series of 8 postterm infants and 4 infants showing the pla-

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cental dysfunction syndrome, all but 2 showed significant lesions of the fetal adrenal cortex. To test the significance of these findings, 138 consecutive autopsies of perinatal deaths were studied. In reviewing the morphologic features of the postterm infants and those with the placental dysfunction syndrome, nothing was observed which could be construed as pathognomonic of either condition. However, degenerative and hemorrhagic lesions of the fetal adrenal cortex occurred with significantly greater frequency in the study group than in a random population subjected to perinatal autopsies. It is possible that these changes might represent precocious involution of the fetal adrenal cortex, having its inception in utero, and these changes may reflect inadequacies of placental function.

Preinvasive Cancer of the Uterine Cervix. E. S. Taylor and L. C. Walker. Postgrad. Med. **23**:105-108 (Feb.) 1958 [Minneapolis].

Between Dec. 1, 1947, and Jan. 1, 1957, 11,324 patients examined in the department of obstetrics and gynecology of the University of Colorado School of Medicine in Denver were subjected to the Papanicolaou vaginal cytological test as a routine screening procedure. Fifty-one of the 11,324 patients screened were detected as having preinvasive carcinoma of the uterine cervix. Twenty additional patients were found to have preinvasive cancer by a primary biopsy. The 4th decade of life accounted for the largest group, rather than the 5th decade, which predominates in cases of invasive cervical cancer. All the patients except 1 were married, and all but 5 had had children. The 1 unmarried patient had been pregnant. This finding is in keeping with the fact that cervical cancer is a disease of the noncelibate. There were relatively few of the classical symptoms usually attributed to early cervical cancer. About 33% of the patients were seen because of pregnancy; another 33% had a menstrual disorder or vaginal discharge; and the remaining 33% had various complaints, none of which suggested early cancer. These findings emphasize that carcinoma in situ is a disease without signs or symptoms and, therefore, can usually be discovered only by Papanicolaou vaginal cytological testing, followed by a 3-stage uterine biopsy taken from the squamocolumnar junction of the cervix, from the endocervix, and from the endometrium, to substantiate or to rule out diagnosis of preinvasive carcinoma or possibly invasive carcinoma.

Fifty-three of the 71 patients received definite treatment, consisting of total abdominal or vaginal hysterectomy with excision of 2 cm. of the upper vagina in 50 and of radium treatment in 3 in whom the general condition contraindicated major surgical treatment. There were no operative deaths, and cancer has not recurred in any of these patients after treatment. The remaining 18 patients had only a cervical conization for reasons of their youth and their desire to have more children. Four of the 18 were lost to follow up, and 1 died of adenocarcinoma of the rectum. Eleven of the remaining 13 patients are free of any biopsy or vaginal cytological evidence of disease. A patient in whom the diagnosis of in situ carcinoma of the cervix has been made has a 44% chance of cure by simple cone biopsy alone.

Cytological Screening for Cervical Cancer: Comparative Findings in a 6-Year Survey of a Well Population. E. Stern. Cancer **11**:122-126 (Jan.-Feb.) 1958 [Philadelphia].

The examination of vaginal smears from 39,387 women, during the years from 1950 to 1955 inclusive, led to the detection of 303 genital cancers. This study was limited to the primary cancers of the uterus and, more particularly, to the 241 squamous cancers of the cervix. The group included all the proved cervical cancers seen at the clinic except for the occasional clinically advanced case in which the diagnosis was obvious and in which smears were not taken because of danger of hemorrhage from manipulation. During the period of survey there was a progressive increase in the number of in situ cases discovered, that is, the increment was almost entirely accounted for by the increased number of cancer lesions in the stage 0 group. The main change in procedure during the 6-year period was the increasing use of the smear as part of the examination, with the resulting increase in discovery of in situ cases, per 1,000 population, from 0.86 in 1950 to 3.0 in 1955. The real increase in effectiveness of this policy is partially concealed by the fact that a sizable proportion of the total group examined each year are individuals who have been examined previously by the clinic. The incidence of 3 in situ cervical cancers per 1,000 population when all individuals were screened is consistent with that statistically expected in the general population. The stage 0 cancer lesions showed a peak incidence when the patients were between 30 and 39 years of age, but they were found at all ages from 20 to 70 years.

PEDIATRICS

Stevens-Johnson Syndrome. D. A. Sher. Minnesota Med. **41**:32-34 (Jan.) 1958 [St. Paul].

The author reports a case of Stevens-Johnson syndrome, a variety of erythema multiforme exudativum, in a 10-year-old girl. Physical examination on admission to the hospital revealed a toxic, dehydrated, and acutely ill child. The temperature was 105.4 F (40.8 C). The pulse was rapid, and restlessness was marked. There was a generalized,

widely spread rash, involving the palms and soles. The mucous membranes of the mouth were ulcerated, and a conjunctivitis was present. The patient was placed on therapy with penicillin, erythromycin (Ilotycin), and parenterally administered fluids. Her condition became progressively worse during the next 2 days. There was a severe conjunctivitis with pericorneal vascular injection and marked purulent drainage. The buccal mucous membrane showed an ulcerated appearance, and considerable sloughing was noted. The tongue was coated and ulcerated. There was a brownish coating on the gums and teeth. The tonsils had a grayish exudate, and the pharynx was injected and swollen. The posterior lymph nodes were palpable. The skin presented a generalized papular, macular, vesicular eruption; large bullae were observed on the face, abdomen, and buttocks. Slight pressure over the lesions caused superficial peeling of the skin, leaving a violet-red raw surface. Penicillin and erythromycin were withdrawn, and the patient was given 20 mg. of corticotropin (ACTH) every 6 hours and 100 mg. of tetracycline (Achromycin) every 8 hours, 10% dextrose by continuous drip, vitamin B complex, and vitamin C. There was an apparent dramatic response to the corticotropin and supportive therapy. Administration of corticotropin and tetracycline was continued for 10 days with a gradual decrease in dosage. The skin lesions and mucous membrane involvement cleared rapidly, and the patient was discharged 15 days after admission to the hospital.

The syndrome is characterized by a variation in skin reaction, involvement of the mucous membranes, severe ophthalmia, and severe general reaction. The causation of the syndrome has not yet been elucidated. The lack of a biological syndrome, the ineffectiveness of antibiotics alone, and the dramatic effects from the administration of corticosteroids seem to indicate an allergic basis for the disease. The disease is self-limiting and variable in its course. Since sensitization may play a role, antihistamines and adrenal steroids are indicated in treatment. Antibiotics, except those with possible sensitizing properties, should be given to control secondary bacterial invaders. Supportive therapy is essential because of the severity of the disease.

Obstructing Subglottic Hemangioma of the Larynx in Infancy: Review of the Literature and Report of a Deceptive Case. P. Doermann, J. Lunseth and R. H. Segnitz. New England J. Med. 258:68-71 (Jan. 9) 1958 [Boston].

A subglottic hemangioma in a 1-month-old female infant proved extremely deceptive and was diagnosed with certainty only at autopsy. Upper respiratory obstruction was a significant and characteristic clinical observation. A unique feature was that the hemangioma was not grossly apparent by direct laryngoscopy or bronchoscopy. Autopsy revealed a symmetrical stenosis of the subglottic lumen without discoloration of the overlying mucosa. The sectioned area revealed spongy, brown tissue in the submucosa, involving approximately the anterior three-fourths of the circumference of the trachea between the vocal cords and the cricoid cartilage. The lesion measured 3 or 4 mm. in thickness and practically occluded the lumen of the subglottic area. Only 14 previous cases of juvenile subglottic hemangioma have been reported. This lesion is probably more prevalent than indicated by reports and should be considered in the differential diagnosis of upper respiratory obstruction in infants. Irradiation preceded by tracheotomy is recommended as the treatment for proved cases and might also be undertaken when the site of respiratory obstruction and the clinical course are suggestive of this deceptive lesion.

Endobronchial Tuberculosis in Children: A Study of 156 Patients. E. M. Lincoln, L. C. Harris, S. Bovornkitti and R. W. Carretero. Am. Rev. Tuberc. 77:39-61 (Jan.) 1958 [New York].

In the chest clinic of the children's medical service of Bellevue Hospital, in which nearly 1,000 consecutive cases of primary pulmonary tuberculosis were observed from 1930 through 1946, a study of endobronchial disease in children with primary pulmonary tuberculosis was begun soon after antituberculous drugs became available. A preliminary report in 1952 showed that the results of specific therapy were disappointing; no evidence was obtained by bronchoscopy that streptomycin had a beneficial effect on endobronchial involvement due to encroachment of tubercular nodes on bronchi. A technique of anesthesia was developed with the purpose of avoiding unnecessary psychic and physical trauma owing to repeated bronchoscopies.

The present report is based on 156 children who had bronchoscopic evidence of tuberculous endobronchitis. Endobronchial tuberculosis was more frequent in children less than 4 years of age and in boys. Severe symptoms were more often observed in young infants. Roentgenologic evidence of obstruction was observed in 90% of the patients. Evidence of segmental obstruction was more commonly seen in the right lung and most often in the right middle lobe and in the anterior segment of the right upper lobe. Seventy-seven per cent of the group received antimicrobial therapy. In 66% of those who were treated, the medication was given for more than 6 months; in 48%, for more than a year.

Granulation tissue was the most frequent finding on bronchoscopy. Fifty-four of the 156 patients had polyps, and erosion of the bronchial wall was diagnosed in 25 patients. The duration of endo-

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bronchial involvement could be measured by bronchoscopy in only 70 patients. It continued for less than 3 months in 11 children, 4 of whom were untreated, and from 1 to more than 3 years in 23 children, 18 of whom received specific therapy, 10 for more than a year. Bronchograms were done on 103 patients without selection and showed abnormal findings in 70 and marked abnormalities in 50. Bronchiectasis was the most common evidence of damage, occurring in 70% of those with marked abnormalities. The percentage of children with abnormal bronchograms was similar in the treated and untreated groups. There is no evidence that antimicrobial therapy markedly shortens the course of tuberculous endobronchitis due to encroachment of caseous nodes on the bronchi. There is also no evidence that the use of specific therapy diminishes the incidence of sequelae in the bronchi and the parenchyma. Nevertheless, there is justification for treating children with tuberculous endobronchitis in the hope of diminishing the dangers of bronchogenic spread of the tuberculosis.

Preparation of Children for Tonsillectomy. H. J. Jones. M. J. Australia 1:32-34 (Jan. 11) 1958 [Sydney].

The effectiveness of preparing a child for hospitalization by the use of a booklet to reduce emotional disturbance after tonsillectomy was investigated by analyzing the behavior of 28 boys, 14 of whom received the booklet. The boys ranged in age from 5 years and 8 months to 6 years and 10 months. The booklet used was similar to several used in the United States. It was entitled "Going to Hospital" and contained 5 line drawings of hospital scenes and 4 pages of text. The account included details of admission procedure, preoperative examination and preparation, induction of and recovery from anesthesia, and the postoperative period in the hospital. A letter was sent with the booklet, suggesting its method of use and recommending that the child should color the illustrations. The behavior of each child was assessed prior to his admission to the hospital, while in the hospital, and for a period of 3 months after his discharge. The booklet seemed to have some effect, but the results were not statistically significant. Every child experienced some emotional reaction, which was mainly centered around changes in the sleep pattern, appetite, dependency, and temperament. These were at their maximum at 1 week, were considerably less at 1 month, and had usually disappeared 3 months after operation.

The investigation illustrated some of the difficulties that are met with in this type of research. As the socioeconomic and educational backgrounds of the mothers differed, so did their reports vary in detail and accuracy. The greatest difficulty was encountered in ascribing numerical quantities to emotional factors and in establishing a base line for each child from which to asseses variations in behavior. From the results of this study several improvements in the booklet could be made. It had been the belief that unpleasant as well as pleasant incidents should be included, so that a child knew that he would get a "prick in the arm" and that this might "hurt a little bit." In view of the finding that 10 of the children were afraid of "needles" beforehand, this reference to injections could well be omitted. All the mothers who received the booklet expressed their enthusiastic approval of its use and reported that their children enjoyed coloring the illustrations.

UROLOGY

A Treatment of Undescended Testicle in Infancy. C. G. Scorer. Arch. Dis. Childhood 32:520-522 (Dec.) 1957 [London].

Of 2,700 boys examined in the maternity department of a general hospital, 108 were found to have incomplete descent of 1 or both testes. In 89 of these the testis was found to descend during the first year of life. The remaining 19 boys included 5 in whom the testes were small and at the external ring, 3 testes had never been felt and were presumably within the inguinal canal or abdomen, and 11 persisted just outside the top of the scrotum. The author is especially concerned with the group of 11 boys who presented the latter condition with 3 distinctive features. 1. The testis was present at birth or appeared within the first few days of life. 2. It was of good size and firm consistency. 3. It did not change position during the many weeks or months of observation; it remained at the pubic crest or, at most, could be pushed 1 to 2 cm. below it. In several of these boys herniation into the unclosed processus vaginalis resulted, and in treating these hernias by surgical excision of the sac, the author found that the pathway from the external ring to the scrotum was blocked by a definite septum, or fibrous barrier, which had to be broken through by firm pressure before the scrotal cavity could be reached. This seemed to indicate that the cause of failure of descent is an obstruction and is not inherent in a deformity of the testis or a shortness of the cord. The rational treatment is, therefore, to remove the obstruction at an early age to enable the testis to descend spontaneously or to place the organ in its normal position. The policy of waiting and the subsequent use of hormones is unlikely ever to be rewarded.

The following operation was performed on the 11 boys. The superficial inguinal pouch is opened by an incision over the external ring. The testis and the cord with their coverings are dissected clear. The tunica vaginalis is opened and explored

with a probe to find out if it communicates with the peritoneal cavity. If a hernia is present, the sac is dissected off the cord and ligated as high as possible. The lower end of the superficial inguinal pouch is then broken through with pressure by the little finger. A stitch of nylon is passed through the gubernaculum, which at this age is a thick and strong structure attached to the lower pole of the testis. Both ends of the nylon are threaded into the inguinal incision and out through the bottom of the scrotum, thus drawing the testis down toward its normal position. After closing the inguinal wound, the stitch is attached to the skin halfway down the inner side of the thigh. The stitch is kept in for 2 or 3 days, and tight napkins are not allowed for several days. Ten of the 11 infants treated now have the testis between 5 and 7 cm. from the pubic crest. The testis could not always descend fully because the scrotum was often smaller. Age at operation varied between 3 and 52 weeks, but the majority of the infants were under 3 months, and this appears to be the most satisfactory time.

OPHTHALMOLOGY

The Early Detection of Glaucoma: Results of 2,000 Routine Tonometries. H. Reed and J. E. L. Bendor-Samuel. Canad. M. A. J. 78:6-10 (Jan. 1) 1958 [Toronto].

The authors comment on the results of routine tonometry in 2,000 patients over 40 years of age. In 28 of these patients glaucoma had been diagnosed on the basis of the history and the clinical examination before tonometry was performed. Tonometry revealed raised intraocular tension in an additional 71 patients who appeared clinically normal. In 30 of these patients additional tests indicated the presence of glaucoma; in 13 the investigations were negative; 7 are still being followed up as doubtful; and 21 failed to attend for investigation. The authors consider it likely that most of the 7 doubtful patients will later show definite signs of the disease. Thus, about 2% of the patients over the age of 40 had early glaucoma without symptoms or obvious clinical signs.

Of the 30 patients who were believed to have glaucoma, only 6 patients had narrow angles. It is a common observation that there are relatively more wide-angle glaucomas than narrow-angle glaucomas. Five patients had field defects. The water test was performed in 16 of the 30 patients and proved positive in 10 of them; the mydriatic test was positive in 3 of 4 patients. These provocative tests will often prove conclusively that glaucoma is present; a negative result, however, does not necessarily exclude the disease. Tonography proved of great value both in diagnosis and in assessing the effectiveness of miotics. Routine tonometry by ophthalmologists is only part of the answer to this problem of unrecognized glaucoma. General physicians should be urged to include tonometry as part of every routine physical examination of patients over the age of 40. If the ocular tension is raised, the patient should be referred to an ophthalmologist for investigation. Medical schools should teach this simple test to the physicians of tomorrow.

Ocular Signs of Meningioma. F. W. Newell and T. C. Beaman. Am. J. Ophth. 45:31-40 (Jan.) 1958 [Chicago].

The records of 143 patients with histologically verified intracranial meningiomas were reviewed with special reference to the ocular signs and to the fact that, if the meningiomas are removed early in their course, there may be a return to normal neural function. There were 14 patients with meningioma of the tuberculum sellae, 11 of whom had optic atrophy with vision in 1 or both eyes reduced to less than counting fingers at 2 ft. In 2 of these patients roentgenograms of the skull and contrast radiography did not indicate the abnormality, but the tumor was found by surgical exposure. Meningioma should be thought of as a likely cause of reduced vision in middle-aged patients with progressive optic atrophy not arising from glaucoma, vascular disease, or syphilis. Meningioma of the sphenoidal ridge was found in 15 patients. Symptoms varied with the portion of the ridge involved, but optic atrophy of the homolateral eye and proptosis were the outstanding signs. Roentgen-ray examination will frequently aid in the diagnosis, since meningiomas in this area, particularly those involving the pterional portion of the sphenoidal ridge, cause a characteristic hyperostosis.

The optic nerve within the orbit may be involved in a meningioma that originates within the cranium or at the optic foramen or within the orbit itself. The chief symptoms of optic nerve meningioma are decreased vision and proptosis which are slowly progressive. Either may occur without the other. Two patients with meningioma extending along the optic nerve sheath were seen in this group. Proptosis was the outstanding symptom in the first, and reduction of vision in the second. The reports of patients with meningioma extending along the optic nerve sheaths, together with the findings in these 2 patients, indicate the futility of purely ocular surgery in their management. The removal of such tumors is the province of the neurosurgeon who can unroof the orbit and inspect the optic foramen. Tumors of the olfactory groove attain a large size before causing visual symptoms. Of 10 patients with meningiomas in this area, the majority had no ocular signs, and the visual signs that did occur aided in localizing the tumor in only 1 instance.

INDUSTRIAL MEDICINE

Hearing Impairment from Noise of Pneumatic Drills. L. Risch. J. Laryng. & Otol. 71:846-849 (Dec.) 1957 [London].

The author tested the hearing of 9 pneumatic-drill operators engaged in road repair work. Their ages ranged from 30 to 55 years. There was an interval of a few minutes between the end of drilling and the test, which was carried out in a quiet, acoustically treated room. The men were questioned concerning their experiences connected with drilling and especially concerning tinnitus and hearing of speech. The intensity of the noise produced by the drill was measured. At a distance of 6 ft. the noise from a single drill had an intensity of 95 db. Probably the intensity is higher between 2 machines, when several drills are used simultaneously in a team, but measuring this was not feasible. While this was too small a group for far-reaching deductions, the author believes that the following conclusions can be made: It is possible for some individuals to be exposed for many hours a day to very loud noise (at least 95 db.) for several years without necessarily suffering serious damage to the function of hearing. Three of the tested men worked with pneumatic drills for 10 years, and in 2 (1 started at the age of 20 and the other at 25) the hearing was only slightly impaired for the highest frequencies. In the third man, who was 55 years old, there was a more marked impairment in 1 ear. In the majority of pneumatic-drill operators the noise does impair the hearing, and in a few the impairment can be of such a degree that hearing for speech in everyday life can be seriously affected. In severe unilateral deafness from other causes, such as was discovered in 1 of the men tested, even a moderate impairment for the higher notes in the good ear is bound to have a serious effect on hearing speech. Such a person should be discouraged from undertaking this type of work. The comparatively slight impairment of hearing in the majority, in spite of exposure to this severe and unpleasant noise, indicates that the use of proper earplugs could prevent damage to the ears from this particular type of industrial noise.

Roentgen Resurvey of Cement Workers. O. A. Sander. A. M. A. Arch. Indust. Health. 17:96-103 (Feb.) 1958 [Chicago].

One hundred ninety-five cement workers who had had from 20 to 45 years of exposure to raw, mixed, or finished cement dust were studied by comparing their 1934 and 1937 chest roentgenograms with recent ones. One hundred twenty of the 195 cement workers (or 61.5%) were classified as having normal chest roentgenograms; the films of 68 (or 35%) showed "moderate linear exaggeration"; the films of 6 (or 3%) showed "marked linear exaggeration with ill-defined micronodular shadows." Because of the small size of these round shadows (not over 1 mm.) and because of the normal appearance of the root shadows, these 6 cases do not resemble silicosis and cannot be so diagnosed. Only 1 of the 195 patients showed clear-cut micronodulation, the exact cause of which could not be determined.

It is suggested that the linear exaggerations and the micronodulations appearing on the roentgenograms are due to retention of some of the dust in the lymphatics of the lungs. These changes did not resemble silicosis in any case because of the lack of enlargement of the root lymph nodes, and it is suggested that they may be due to direct shadows cast by focal collections of retained dust and not by fibrosis. Exposure to finished cement dust alone results in no recognizable x-ray changes even after upwards of 30 years of exposure, indicating that finished cement is largely absorbed and that there is little or no retention and no resulting fibrosis. Inhalation of raw, mixed, or finished cement dust does not predispose to tuberculosis or emphysema.

THERAPEUTICS

Antituberculous Chemoprophylaxis with Isoniazid: Preliminary Note. A. O. Zorini. Dis. Chest. 33:1-17 (Jan.) 1958 [Chicago].

The author administered isoniazid to 600 children between the ages of 4 and 11 years. These children came from tuberculous families, most of whom had positive reactions to the tuberculin skin test but did not have clinical or radiologic manifestations of active tuberculosis. Radiologic examination of the children showed signs of progressive infection in the forms of hilar calcification, parenchymal calcific foci, enlargement of hilar shadows without perifocal reaction, and alteration of the bronchovascular markings. Circulating antibody determination was normal in most children. The erythrocyte sedimentation rate was normal. Slight eosinophilia was noted in some. Special preparations, such as fruit jelly and chocolates containing 50 and 100 mg. of isoniazid and biscuits containing 50 mg. of isoniazid, were given to small children, while isoniazid in tablets or syrups was given to older children. The drug was administered in an initial daily dose of 10 mg. per kilogram of body weight, followed by an increment of 5 mg. per kilogram of body weight daily each week, until a dose of 20 mg. per kilogram of body weight daily was attained. The daily dose did not exceed 800 mg. in any of the children. Fifty per cent of the children received chemoprophylaxis for 4 months, and the other 50% for 2 months. The drug tolerance was excellent in nearly all the children with regard to both daily and total doses. Discontinuation of

treatment was necessary in only 4 of the 600 children, because of a relapsing dermatitis in 2 and because of gastroenteritis in 2.

The clinical improvement in nearly all the children was featured by increase in body weight. In the 100 children followed by controlled intradermal testing, a nearly equal number of increased, stationary, or decreased degrees of reactivity were noted. Negative reactions were not observed. These data show that prophylactic treatment with high doses of isoniazid can be carried out without untoward reactions in tuberculin-positive or tuberculin-negative persons who do not have specific, active pulmonary lesions. The author is convinced that simple isoniazid chemoprophylaxis is capable of exerting immediate therapeutic action against existent tubercular foci within lymph nodes and other organs and will furnish through the isoniazid-resistant mechanism or some other mechanism a greater resistance against an endogenous or an exogenous reinfection. It is important to repeat isoniazid treatment at appropriate intervals during the second and the third year, especially in infants born of tuberculous parents. Canetti's proposal of combining chemoprophylaxis with a BCG strain vaccination, resistant to isoniazid and catalase-positive, would be difficult to put into practice and would carry with it a certain danger. It should, however, be given tentative consideration in those countries in which BCG vaccination is widely used and psychologically accepted.

Short-term Penicillin and Dihydrostreptomycin Therapy of Streptococcal Endocarditis: Results of the Treatment of 35 Patients. R. Tompsett, W. C. Robbins and C. Berntsen Jr. Am. J. Med. 24:57-67 (Jan.) 1958 [New York].

Seventeen men and 18 women, between 17 and 67 years of age, with endocarditis caused by penicillin-susceptible streptococci, were given a 14-day course of concurrent administration of 500,000 units of potassium penicillin G intramuscularly every 2 hours (a total daily dose of 6 million units) and 0.5 Gm. of dihydrostreptomycin 4 times a day or 1 Gm. twice a day intramuscularly (a total daily dose of 2 Gm.). After the completion of treatment blood cultures were usually taken 3 times a week for the ensuing 2 weeks and at longer intervals thereafter for at least 3 months. Definite bacteriological cure was achieved in 24 patients (69%) and probable bacteriological cure in an additional 8 patients (23%). Bacteriological relapse was observed in 3 patients (about 10%), 1 of whom relapsed twice.

Concurrent administration of penicillin and dihydrostreptomycin probably represents the most satisfactory bactericidal, antimicrobial therapy available today for infections caused by strains of streptococci which are sensitive to penicillin in in vitro tests. The use of this therapy, however, for a period of as short as 14 days falls short of the goal of antimicrobial therapy, which is bacteriological cure in all cases. As it is impossible to predict which patients will not achieve cure with short-term therapy, it may be advisable to extend the routine therapy of endocarditis caused by streptococci for an additional 7 to 14 days beyond the initial 2 weeks, in the hope that the remaining 10% of the patients will be cured by the first course of therapy. Valid theoretical as well as statistical reasons are adduced to support this advice. There was no significant change in the in vitro sensitivity to either penicillin or streptomycin in the strains isolated after unsuccessful short-term combined therapy, indicating that concurrent administration of the 2 antibiotics prevented the emergence of streptomycin resistance.

PATHOLOGY

Carcinoids. G. Nese. Tidsskr. norske lægefor. 77: 947-952 (Nov. 1) 1957 (In Norwegian) [Oslo].

Report is presented of 55 cases of carcinoids, in 21 men and 34 women, from the Institute of General and Experimental Pathology in the Rikshospital and from Kreyberg's personal material. The youngest patient was 11 years old, the oldest was 80; 26 patients were under 30. Of the 13 malignant carcinoids, in 10 men and 3 women, 1 was in the appendix, 4 were in the ileum, 6 were ileocecal, 1 was in the ascending colon, and 1 was in the stomach. Five of the men and 1 woman died. None of the 42 patients with benign carcinoids have died, but 1 patient had a recurrence and was reoperated on. The longest follow-up period for benign carcinoids was 24 years and for malignant carcinoids, 8 years. Carcinoids in the appendix, usually benign, occur most often in young women. Carcinoids in other regions occur far more often in older patients, mostly men. Benign carcinoids may become malignant after some years, but they generally remain unchanged for decades. Treatment is operative. The prognosis for patients with benign carcinoids is good when the tumor is removed, and it is not wholly bad for patients with metastasizing carcinoids if the primary tumor is removed. In 4 patients with malignant carcinoids and metastases, examination of the urine gave evidence of an increased serotonin content.

Further Studies on the Transmission of Staph. Aureus. R. Hare and M. Ridley. Brit. M. J. 1:69-73 (Jan. 11) 1958 [London].

The authors made extensive surveys of the skin and clothing of 12 men and 3 women who were nasal carriers of Staphylococcus pyogenes var. aureus. Isolation of Staph. aureus was carried out

on nutrient-agar plates containing phenolphthalein diphosphate. Six of the 15 carriers had much less contamination of the skin or clothing than the remaining 9. The latter had Staph. aureus sometimes in considerable numbers on the skin and usually on sites having direct contact with the nose, i. e., the face, palms, and fingers. Forearms and wrists were seldom contaminated. The hair, chest, abdomen, back, and legs were either free or the numbers of Staph. aureus were small. The handkerchief, the only item of clothing likely to have direct contact with the nose, was usually heavily contaminated. So, too, was the pocket in which the handkerchief was kept, but staphylococci could also be found on one or another site on the remainder of the clothing and certainly on that of the front of the body. Contrary to expectation, there was no correlation between the number of Staph. aureus in the nose and the intensity and extent of contamination of the skin or clothing. Nevertheless, if the carriers studied be considered representative of carriers in general, about 60% have sufficient numbers of these bacteria on various sites of the skin and clothing to enable them to transmit their own organisms to other persons with whom they come into direct physical contact by means of objects, such as blankets or towels which they have touched, or as a result of dispersal into free air during movement.

Widespread contamination of 1 of the 12 male carriers, who was a healthy person with no pyogenic or other lesions of the skin and only a few Staph. aureus of a different phage pattern in 1 nostril, made it appear probable that the organisms were derived from some situation other than his nose, where there was sufficient warmth and moisture to enable them to multiply. In this individual large numbers of the organisms were obtained from the perineum. However, there was so heavy a growth of coagulase-negative skin staphylococci from the latter area that, without the assitance afforded by the phenolphthalein diphosphate medium, the colonies of Staph. aureus might have been missed. The clothing in apposition to the perineum, such as the legs and seat of the drawers, was heavily contaminated. There was no evidence that the perineum had been contaminated by Staph. aureus coming from the feces. It seemed that the perineum of this particular carrier had been the primary source of the Staph. aureus on his skin and clothing all along, and that this species was able to multiply on this area of the body. Organisms from this reservoir were able to reach other sites on the skin and clothing and be dispersed during movement to much the same extent as with nasal carriers. Thus, when possible sources of infection are being sought, the existence of nonnasal carriers of Staph. aureus must not be overlooked.

RADIOLOGY

Clinical Impressions in 100 Consecutive Cases of Carcinoma of the Urinary Bladder Treated by Supervoltage. C. A. Cuccia, S. Jones and C. M. Crigler. J. Urol. 79:99-109 (Jan.) 1958 [Baltimore].

All patients with carcinoma of the bladder seen in the urology department at the M. D. Anderson Hospital, Houston, Texas, were evaluated with the radiotherapy department for disposition. The emphasis since April, 1954, has been directed toward radical irradiation with supervoltage. Since this program was started, external irradiation with supervoltage has been used in some lesions which otherwise could have been treated with interstitial or intracavitary irradiation. The aim was to treat the empty whole bladder rather than part of it. This decision to treat the whole bladder and to prefer external irradiation was based on Roger Baker's demonstration of circumferential lymphatic spread of cancer cells along the muscle wall. Data are presented on 100 patients with cancer of the bladder treated with supervoltage from April, 1954, to September, 1956. All were treated with either the radio cobalt (Co^{60}) irradiator or the betatron.

In the first year the patients were alternated between the 2 machines, with the aim of finding out whether there were differences in the tolerance, reactions, and immediate results. When these factors were found to be fairly equal, the use of the betatron was preferred because of better volume distribution and because it avoided the late fibrosis of subcutaneous tissue in the suprapubic region which sometimes resulted from treatment with Co^{60}. The use of Co^{60} is now reserved almost exclusively for patients with involvement of the anterior wall of the bladder with extension into the prevesical space. A minimal tumor dose of 6,000 r was applied to the whole bladder in 5 weeks with a 3-field arrangement. The fields, 1 anterior in the suprapubic region and 2 posterior at the level of the sacroiliac joints, range in size from 6 by 8 cm. to 8 by 10 cm. By using different sizes of portals and different degrees of angulation of the posterior fields, the primary tumor and the perivesical extensions were adequately treated. On the betatron, the fields were loaded with a ratio 2:1 in favor of the anterior portal. This spares the rectum to some extent. In some patients who had previous suprapubic bladder surgery, the tumor dose was reduced to 5,500 r.

The impressions gained so far indicate that patients with disease extending to the pelvic wall and those with less extensive disease but in poor general condition are better treated with a palliative technique, because those treated with high doses had poor tolerance and more complications. Previous irradiation of any type is a limiting factor. There appeared to be a relationship between recent

suprapubic bladder surgery (especially partial cystectomy) and contracted bladder after irradiation. Presence of bladder neck obstruction or severe cystitis decreased the local tolerance for irradiation. Urinary diversion prior to irradiation may be essential in some cases. Women, especially those in the older age groups, tolerated the treatment less satisfactorily. Surgery demonstrated that irradiated tissue has diminished healing capacity. The analysis of those with superficial low-grade lesions revealed that the potentiality of the bladder mucosa to form new lesions is not eradicated by irradiation. Therefore, transurethral surgery is best for low-grade, low-stage lesions. Recurrences that reveal advancement in stage and/or in grade or lesions that cannot be adequately removed transurethrally can be eradicated by irradiation. High-grade lesions, including undifferentiated and squamous carcinoma and lesions in stages B_2 and C, should be treated radically. Supervoltage therapy in this group was encouraging, and it appears to have a definite place in the management of carcinoma of the urinary bladder.

PUBLIC HEALTH

Chronic Toxicity of 3-(*p*-Chlorophenyl)-1, 1-Dimethylurea (Monuron). H. C. Hodge, E. A. Maynard, W. L. Downs and R. D. Coye. A. M. A. Arch. Indust. Health 17:45-47 (Jan.) 1958 [Chicago].

Monuron [3-(*p*-chlorophenyl)-1, 1-dimethylurea] has been found to be an effective herbicide for the control of many annual and perennial grasses and herbaceous weeds. When applied at rates of 16 to 64 lb. per acre, it has been found to prevent growth of nearly all vegetation for a period of many months. This control of vegetative growth has proved useful in such areas as highway right-of-ways, railroad yards and right-of-ways, lumber yards, petroleum tank farms, drainage ditches, and industrial plant sites. When used at a much lower application rate, from 1 to 6 lb. per acre, Monuron has proved valuable in selective weed control among crops, such as asparagus, citrus fruits, cotton, grapes, onions, pineapples, spinach, and sugar cane. In recognition of the possible exposure of humans and animals to Monuron, extensive chronic toxicity tests were completed prior to establishment of the residue tolerances. This report describes the results of feeding Monuron for 2 years to rats and for 1 years to dogs.

In these studies 25 mg. of Monuron per kilogram of body weight has been shown to be a noneffective dose even on long-continued administration. Approximately 10 times this dose was associated with growth retardation, a slight anemia, and liver and spleen weight increases (in female rats). The noneffective level, if expressed as the concentration in the diet to give the same dose for rats and dogs, falls in the range of 250-500 ppm of the diet. This concentration range exceeds by 35 to 70 times the residue tolerance figure for asparagus and by 250 to 500 times the residue tolerance figure for most crops. If man is no more susceptible to the toxic effects of Monuron than are rats and dogs, and assuming that only a minor fraction of the human diet will contain Monuron, the margin of safety in the established residue tolerances seems adequate.

Care of Children with Nephrosis and Cystic Fibrosis of the Pancreas in a Crippled Children Program. S. G. Dodd and V. Shannon. Am. J. Pub. Health 48:15-21 (Jan.) 1958 [Albany, N. Y.].

Children suffering from nephrosis and cystic fibrosis of the pancreas have recently been included in the Connecticut State Department of Health's program for crippled children. The authors believe that the inclusion of such children is appropriate, as these diseases are amenable to medical management, but they are catastrophic to the affected individuals and families and frequently require the use of resources beyond those of the family. Connecticut is the first state to take on these new aspects of crippled children's care. The services available under the nephrosis and cystic fibrosis programs are similar to those given to children with other crippling conditions. The programs follow the purposes set forth by the Children's Bureau: to provide skilled medical, surgical, nursing, social, and physical therapy services for children in hospitals, convalescent homes, foster homes, and in their own homes, and to cooperate with other resources in providing and promoting the development of services to crippled children.

Children up to 21 years of age who reside in the state are eligible for diagnostic service. Eligibility for treatment is determined by several factors, including diagnosis, lack of other treatment facilities, need for a planned program of medical care rather than provision of an isolated treatment, and the economic situation of the family. The programs may assist in care of children at home by providing medical consultation, laboratory and x-ray work, and some medication. Funds are also available for hospital and convalescent care. Depending upon the need, continuing medical, social, nutritional, and public health nursing services are provided. The parents are encouraged to maintain close touch with their family physician at all times, and arrangements are made with his foreknowledge and approval. For a child to receive aid, a referral is usually made by the family physician or pediatrician. If the family contacts the department first, they are requested to give their physician's name, and permission to confer with him is obtained. Case histories are cited to explain how the programs function.

BOOK REVIEWS

Introduction to Anesthesia: The Principles of Safe Practice. By Robert D. Dripps, M.D., Professor and Chairman, Department of Anesthesiology, Schools of Medicine, University of Pennsylvania, Philadelphia, James E. Eckenhoff, M.D., Professor of Anesthesiology, Schools of Medicine, University of Pennsylvania, and Leroy D. Vandam, M.D., Clinical Professor of Anesthesia, Harvard Medical School, Boston. Cloth. $4.75. Pp. 266, with 47 illustrations, line drawings by Leroy D. Vandam. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W. C. 2, England, 1957.

The authors of this work, which originally was privately printed in 1949 for use in the department of anesthesiology of the Hospital of the University of Pennsylvania, are well known. They have covered the field of anesthesiology rather well and have added a section on the management of narcotic poisoning. They have limited themselves to few words, a practice which calls for great accuracy of expression. This third edition is the first which has been made available to the profession generally. It is printed on good paper, is easily read, and is fairly well indexed. It is a pleasure to find a book as well done as this one. Anyone who is interested in anthesiology will probably want to own it.

The Lower Urinary Tract in Childhood: Some Correlated Clinical and Roentgenologic Observations. By Sven Roland Kjellberg, Nils Olof Ericsson, and Ulf Rudhe. Cloth. $18.50. Pp. 298, with 265 illustrations. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11; Almqvist & Wiksells, 26 Gamla Brogatan, Stockholm C; Box 47, Uppsala, Sweden, 1957.

In this unusual book the subject matter measures up to the high standard of investigative work that has come to be the expected as routine from Scandinavia. The department of roentgenology at the Karolinska Sjukhuset, Stockholm, has always pioneered in new types of investigation. Some have thought that many of these roentgenologic examinations were superfluous, in that the information could be obtained more easily by other methods, but it must be admitted that persistence has illuminated many obscure problems. Micturition cystourethrography, which forms the basis of this book, falls into this category. The conslusions reached are sound. The book is more than a treatise on voiding cystourethrography in infants and children; it is also a comprehensive summary of current opinion of various pediatric-urologic problems, including an excellent bibliography.

The first chapter is a well-written treatise on the embryology of the genitourinary tract, stressing the important failures in development that result in the more commonly encountered anomalies. In chapter 3, the authors describe their ingenious apparatus for making simultaneous lateral and frontal views with the child sitting in a specially designed canvas swing. The cystographic medium used will also surprise most American urologists, as it consists of a thin solution of barium sulfate (250 Gm. in a 1,000-cc. mixture). The authors have found that, in contrast to mediums containing iodine, it is nonirritating; thus, the child has no discomfort and can be induced to void without much trouble. Films are exposed during the act of micturition. The authors first present films to demonstrate the appearance of the normal bladder and urethra. Although the frontal views will be readily interpreted by the reader, interpretation of the lateral views will require some study. The rest of the book is devoted to the use of the voiding cystourethrogram in the following conditions: ectopic ureter and urethrocele, the neurogenic bladder, bladder neck obstruction, congenital urethral valves, urinary tract infection, stricture of the urethra, intersexuality, and enuresis.

Most urologists believe that cystography has little place in the diagnosis of ectopic ureters and ureterocele. The authors demonstrate that the voiding cystourethrogram is most helpful in demonstrating large ureteroceles lying close to the vesical neck or just inside the urethra that may be pushed aside or collapsed by the passage of a cystoscope and thereby overlooked. They also show with what astounding frequency an ectopic ureter which opens into the urethra can be filled and visualized with contrast medium as the patient voids. This should be a distinct contribution to diagnosis in such cases, because these orifices are notoriously difficult to visualize and catheterize cystoscopically. Another new concept is the demonstration that spasticity of the external sphincter in neurogenic disease and urinary infection may often be demonstrated by the voiding cystourethrogram but not by the retrograde method. One of the best demonstrations of the value of the voiding cystourethrogram is in the case of congenital urethral values. This has always been a most difficult diagnosis to make cystoscopically, and many cases are overlooked. The authors present films and statistics showing that their method is almost 100% accurate. They admit that in some cases it may be a little difficult to differentiate a valve from a spastic external sphincter but that with moderate experience

These book reviews have been prepared by competent authorities but do not represent the opinions of any medical or other organization unless specifically so stated.

roentgenologic diagnosis is accurate. This book should be read by all urologists and roentgenologists.

An Introduction to Medical Mycology. By George M. Lewis, M.D., Professor of Clinical Medicine (Dermatology), Cornell University Medical College, New York, Mary E. Hopper, M.S., J. Walter Wilson, M.D., Clinical Professor of Medicine, University of Southern California, Los Angeles, and Orda A. Plunkett, Ph.D., Professor of Botany, University of California at Los Angeles. Fourth edition. Cloth. $15. Pp. 453, with illustrations. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1958.

The new edition of this volume, which for 18 years has been a standard textbook in medical and, in particular, dermatologic mycology, represents a significant improvement over the preceding editions both from the practical and the theoretical viewpoints. Drs. Wilson and Plunkett, as co-authors, have rewritten the sections dealing with deep mycoses and contaminants respectively. The nomenclature has been brought up to date, and the results of some of the newer experiments have been incorporated. Descriptions of the culture biopsies of many of the dermatophytes are included. This volume is an introduction to rather than a treatise on medical mycology. This explains the fact that no attempt is made to discuss or weigh the evidence for and against controversial subjects. It might have been preferable to consider black and white piedra separately. Trichophyton concentricum, which causes tinea imbricata, is separated from the other members of the genus Trichophyton, probably because of the difference in the clinical disease produced. It was a fortunate decision on the part of the authors to combine the clinical and laboratory phases of the various mycoses instead of separating them as in previous editions. The illustrations are excellent and much more numerous than in the previous editions. This text can be highly recommended for students and practitioners of medical mycology.

Operative Obstetrics. By R. Gordon Douglas, Professor of Obstetrics and Gynecology, Cornell University Medical College, New York, and William B. Stromme, Attending Obstetrician and Gynecologist, Northwestern Hospital, Minneapolis. Foreword by Nicholson J. Eastman. Cloth. $20. Pp. 735, with illustrations. Appleton-Century-Crofts, Inc., 35 W. 32nd St., New York 1, 1957.

The thoroughness, conciseness, and clarity with which the authors have presented their subject marks this book as a library necessity. It fills a long felt need, both for specialists and for teaching residents. The profusion and appropriateness of the pictures, many times with sketches to supplement the photographs, indicate the thought that went into this valuable book. The section on forceps delivery is particularly well done. The rationale for the use, not only of the forceps but also of the type of forceps to be used is clearly explained. The chapters on preoperative, operative, and postoperative care cover a wealth of material. The authors also discuss the management of the complications of pregnancy, both local and systemic, the indications for anesthesia, and the indications and contraindications for many other procedures. Sound judgment is shown throughout this much-needed textbook.

Pediatric Roentgenology. By Dr. M. A. Lassrich, Prof. Dr. R. Prévôt, and Prof. Dr. K. H. Schäfer. Edited by Prof. Dr. K. H. Schäfer. Translation from German provided by James T. Case, M.D., D.M.R.E., Director, Memorial Cancer Foundation, Santa Barbara, Calif. Cloth. $28. Pp. 333, with 700 illustrations. Grune & Stratton, Inc., 381 Fourth Ave., New York 16; 99 Great Russell St., London, W. C. 1, England, 1957.

This book represents the first appearance in German of a treatise dealing with pediatric roentgenology in more than 20 years. The subject matter and illustrations are presented in atlas form, but the legends, although concise, contain the essential clinical and roentgenologic data. The introductory remarks at the beginning of each chapter adequately cover the technical details. Emphasis has been placed on the subjects of practical importance, concentrating attention on the thoracic and abdominal organs, but the other structures, such as the skeletal system, have not been neglected. The illustrations are beautiful and well selected. The reproductions of roentgenograms are in the positive phase. Where indicated, explanatory schematic drawings have been used to good advantage. There is no bibliography. Dr. Case is to be congratulated for the excellent translation of this book into easily readible form, transferring the thoughts and ideas of the authors accurately. This book is highly recommended for radiologists, pediatricians, and all physicians interested in the diseases of children.

Abdominal Operations by the Vaginal Route. By Paul Werner, M.D., and Julius Sederl, M.D. Translated by L. M. Szamek, M.D. With foreword by Richard W. Te Linde, M.D. Cloth. $9. Pp. 165, with 120 illustrations. J. B. Lippincott Company, 227-231 S. Sixth St., Philadelphia 5; 4865 Western Ave., Montreal 6, Canada; Pitman Medical Publishing Company, Ltd., 45 New Oxford St., London, W. C. 1, England, 1958.

This book is an excellent translation of the one published by the authors in 1952. This does not diminish its quality or value. Dr. Werner continued to perform intricate vaginal operations almost until the time of his death. He was one of the foremost operators, not only in Europe but in the world. His vast experience as a teacher and master technician is evident in the beautiful and instructive drawings. Even the German edition was most instructive for those who could not read German, because of these illustrations. The price of the German edition was $11. The English translation is only $9. It should prove helpful to any surgeon or gynecologist who is interested in perfecting his technique of vaginal operations.

QUESTIONS AND ANSWERS

NOISES IN THE HEAD

To the Editor:—*Some patients complain of ringing or buzzing in the head. This is definitely not described as a patient usually describes a tinnitus, in that it is not lateralized and, when specifically asked regarding whether the sound or sensation is coming from the ears, the answer invariably is, "No, it is in my head." These patients describe this as being continually present day and night, never particularly interfering with sleep but definitely disturbing them during the day. The patient may indicate that for a brief period of time it may suddenly leave and return just as suddenly. Most of these subjects claim that it has been present for years. Examination by otolaryngologists, complete neurological examinations, general physical examinations, and the duration of these symptoms fail to reveal the cause. The patients are not helped by sedatives, tranquilizers, or vasoconstrictors, such as ergotamine tartrate and caffeine. Reassurances to the patient as to the absence of any discernible organic disease and referrals to psychiatrists likewise fail to help. What is the cause of this distressing phenomenon?* *M.D., Washington.*

Answer.—The problem of buzzing, ringing, or humming in the head in a person with no neurological sign, abnormal otological or aural finding, obvious vascular disease, or hypertension presents a vexing problem. One should consider a large aneurysm of the cartoid artery or the circle of Willis, an arteriovenous aneurysm, a variant of labyrinthine hydrops, preternatural pulsation frequently heard at night in people with much arteriosclerosis, with loud cardiac murmurs, or with a sound-producing mechanism elsewhere in the body, spasmodic repetitive contraction of the stapedius muscle, and the possibility of serious mental aberration with neurosis or with hallucinations.

Answer.—The condition described is fairly common among tense or introspective persons. It is probably an exaggeration of the sound normally heard in a sound-proof room. This can be verified by anyone if he gives his attention to it. If those who complain of the sounds described have their attention distracted from them, or if they are asleep, the sounds are not heard. It is therefore considered a functional phenomenon arising from awareness and magnification of a normal occurrence.

RECURRENT AXILLARY AND INGUINAL ADENITIS

To the Editor:—*A 43-year-old man has noted blood and pus in his stools for one year. He has also had recurrent axillary and inguinal adenitis, which has never suppurated. Sigmoidoscopy and barium enema have revealed a cauliflower granulomatous lesion involving the rectal ampulla and 3 or 4 cm. of the rectum extending immediately above the anal canal. There is no distinct stricture or fistula, and the sigmoidoscope can be passed into the sigmoid, revealing a normal mucosa above. The serum albumin is 2.5 Gm. per 100 cc., and the globulin is 5.3 Gm. per 100 cc. Physical examination is negative except for the adenopathy, with nodes of 1 to 1.4 cm. in diameter which are firm, mobile, and nonindurated. Chest x-ray and serologic examination are normal. Rectal biopsy (four separate specimens) revealed chronic inflammation without giant cells. A Frei test was negative at 48 hours, but a distinct nodule was seen at the test site 10 days later. 1. Is venereal lymphogranuloma in this patient likely? Are strictures necessary for such a diagnosis in the male? Is such an infection likely to be the result of homosexual practice? 2. Does the Frei test antigen lose its potency even though the expiration date is given as March, 1958? 3. Is the complement fixation titer affected by performance of the test? 4. Are there any special isolation techniques for identification of the virus from an inguinal node? How should the biopsy specimen be handled? 5. Does sarcoid produce this type of lesion? Must any other granulomatous process be considered? 6. Would liver biopsy aid in establishing the diagnosis? Has the liver been shown to be involved in venereal lymphogranuloma? 7. What is the accepted form of therapy of this disease?*

Leonard Breslaw, M.D., Los Angeles, Calif.

Answer.—1. The findings in this case are diagnostic of venereal lymphogranuloma. Note the reversion of the albumin-globulin ratio of the serum proteins and the positive intracutaneous Frei test in the form of a distinct nodule seen at the site 10 days later. This evidence would outweigh the "negative Frei test" reported. Nodule formation is as important as an erythematous reaction at the site of

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any medical or other organization unless specifically so stated in the reply. Anonymous communications cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

injection. The presence of strictures is not essential to the diagnosis. Aberrant sexual acts (anal coitus) are not the only means of development of this disease. 2. The Frei test antigen does not lose potency for at least two years. 3. The complement fixation titer is not affected by the intracutaneous Frei test. 4. Animal transfers (yolk sac from chick embryo and mouse brain) are the special techniques for the identification of the virus. There is nothing special about the surgical removal of a lymph node for microscopic studies (Kornblith, *Surg. Gynec. & Obst.* 63:99, 1936). 5. Sarcoid does not histologically resemble the lesion under discussion. The only other granulomatous process is, possibly, foreign body granulomas. 6. Biopsy of the liver would not aid in the diagnosis. The liver is not involved in venereal lymphogranuloma. 7. Ordinarily, a course of orally administered broad-spectrum antibiotics with short courses of large doses of sulfonamides should be given for about two months, but, since cancer is so frequently associated with or superimposed on venereal lymphogranuloma, the antibiotic and chemotherapeutic therapy in maximum tolerable dosages should be limited to only two weeks. If no recession of the rectal lesion is observed, local excision of the entire lesion (total biopsy) should be performed and followed by immediate frozen section microscopic examination. If cancer is discovered, an abdominoperineal resection should be then carried out. If not, antibiotic therapy should be continued for many weeks after operation.

MONTHLY SUICIDE RATES

To the Editor:—*The county coroner has noted a great increase in the suicide rate in this area from the early part of December until the middle of January in each of the last four years. Is this in conformity with the national findings? Are there any reasons for this apparent increase during this period of the year?*

Robert W. Viehe, M.D., Whitefish, Mont.

Answer.—There are no statistical data available that would cover such an unusual period as "the early part of December until the middle of January." Available statistical data are grouped, on the national level, according to calendar months. In addition, the National Service of Vital Statistics has not as yet published reports for 1956 and 1957.

The deaths due to suicide in the United States from 1951 to 1955, according to monthly distribution, are given in the table. From these figures the following deductions can be made: The number of suicides in the combined two months of January and December, 1952, has declined by 12 from the number recorded for 1951. Figures for 1953 show an increase of 13 over 1952. In January and December of 1954, the number of suicides increased by 50; in the same two months of 1955, the increase is 42 above the previous year. Thus, it seems that there is a continuous increase in the number of suicides in January and December of every year, 1952 through 1955. These figures are misleading, however, if taken alone, inasmuch as the total number of suicides in the entire United States increased in 1953, 1954, and 1955. The increase during these years can be calculated from the table: 308 in 1953, 409 in 1954, and 404 in 1955. The bimonthly averages in these years, on the national level, are significantly higher than the increases during the months of January and December.

With the figures of the average monthly increase and the comparison of these averages to the actual increase in January and December of the corresponding years, a computation can be made which would show that the increase of suicides in the named months is below the average expected. It appears, even without this computation, that the increase in the number of suicides during the months of January and December, 1953 through 1955, does not show any significant deviation from the increase of suicides during any other month, on the national level.

Death Due to Suicide in the United States from 1951 to 1955 According to Monthly Distribution

	1951	1952	1953	1954	1955
Jan.	1,260	1,255	1,311	1,333	1,369
Feb.	1,198	1,214	1,230	1,240	1,224
March	1,343	1,308	1,435	1,436	1,529
April	1,423	1,403	1,444	1,444	1,550
May	1,537	1,442	1,467	1,448	1,518
June	1,391	1,330	1,360	1,458	1,377
July	1,329	1,303	1,334	1,412	1,370
Aug.	1,321	1,224	1,271	1,366	1,403
Sept.	1,276	1,179	1,292	1,357	1,351
Oct.	1,407	1,330	1,388	1,333	1,389
Nov.	1,312	1,207	1,246	1,349	1,357
Dec.	1,278	1,242	1,269	1,266	1,385
Total	15,909	15,567	15,947	16,356	16,769

HEREDITY AND MYOPIA

To the Editor:—*What are the probabilities of errors of refraction in offspring of mates, each of whom have stationary simple myopia of 2-D severity?*

M.D., Michigan.

Answer.—It is fallacious to give genetic prognostication for myopia taking into account the total refraction of the eye (clinical refraction). This would be possible only if the mode of transmission and varying course of each of the following variable refraction components were known: (1) corneal refraction, (2) depth of anterior chamber, (3) refraction of the lens, and (4) axial length of the eye. The total, or clinical refraction, is the combined result of the above four factors. There is a great need for genetic studies on the inheritance of each of these factors. There are a number of pedigrees of clinical refraction showing dominant inheritance. Some pedigrees suggest intermediate inheritance. In some families in which dominant inheritance is exhibited a wide range in the severity of the myopia exists; in others, there is a rather remarkable consistency in the degree of myopia. A

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high myopia tends to exhibit recessive or sex-linked recessive modes of transmission. This question provides no data concerning the presence of myopia in the siblings of the mates or in their respective parents and thus gives insufficient data on which to base a genetic prognostication. If it is assumed, however, for purpose of speculation, that both mates are heterozygous for a dominant gene for myopia, it can be anticipated that 25% of the offspring will be homozygous for the myopic gene, 50% will be heterozygous for the myopic gene, and 25% will be homozygous for the normal gene. Thus, it can be anticipated that the offspring in each pregnancy has three chances in four of being myopic. This implies a dominant gene for myopia capable of being interpreted only in the light of the total clinical refraction and is not specifically dependent on the previously discussed refraction components.

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Sorsby, A.: Genetics of Ophthalmology, St. Louis, C. V. Mosby Company, 1951, p. 88.

VARIED REACTIONS WITH TEST FOR UROBILINOGEN

To the Editor:—*In an office where Ehrlich's aldehyde test for urobilinogen is used routinely, in certain cases instead of albuminuria a milky, orange-colored reaction has been seen when the reagent was added to urine. What causes this?*

Michael H. Berger, M.D., Quito, Ecuador, South America.

Answer.—This and similar phenomena have been noted many times in cases where the urine test is negative for protein but positive for bile. A positive test for bile in urine indicates the presence of bilirubin and is also associated with the excretion of bile salts. When Ehrlich's reagent is added to a bile-positive urine, the strong hydrochloric acid in the reagent converts bile salts to the acids, which are insoluble. If these are present in sufficiently high concentration, they impart a milky appearance to the urine and may even precipitate. Ehrlich's reagent also acts on the bilirubin to produce a brown pigment. The precise shade of color and degree of turbidity resulting from the addition of the reagent depends on such factors as the concentrations of the urinary pigments, bilirubin, bile salts, and urobilinogen. For example, if little urobilinogen and relatively high concentrations of bile salts and bilirubin are present, as in severe obstructive jaundice, the addition of Ehrlich's reagent gives a turbid, brown suspension. On the other hand, if urobilinogen is present in substantial concentration in addition to bilirubin and bile salts, as in diffuse hepatic disease, the red color resulting from the interaction of Ehrlich's reagent with urobilinogen mixes with the yellow-brown color to give a milky, orange-colored suspension. The interfering action of bile on the urobilinogen test may be avoided in the following way. If the urine test is positive for bile, make 8 ml. of urine slightly alkaline with 1 drop of 20% sodium hydroxide solution. Add 2 ml. of 10% barium chloride solution and mix. Then filter and, to a suitable amount of the filtrate, add an equal volume of Ehrlich's reagent.

SPLITTING OF FINGER TIPS

To the Editor:—*A contractor, when obliged to handle lumber for several days, experiences dryness and painful splitting of his finger tips. All the proprietary remedies he has tried have been of no benefit. Can you suggest anything to alleviate this problem? The handling of lumber is an occasional necessity in his work.*

D. J. Mariea, M.D., Fostoria, Ohio.

Answer.—The report submitted suggests that this patient has a contact dermatitis or dermatitis venenata of the tips of the fingers. This may have resulted because of an acquired sensitivity to the lumber he handles. It is possible, also, that there may be associated mechanical factors, such as rubbing of the finger tips on the boards when they are handled, particularly if the surfaces are rough. It is assumed that there are no other skin lesions, but other body areas should be examined, particularly webs of toes, soles, groins, and gluteal clefts. If there is evidence of skin inflammation in any of those areas, the possibility of trichophytosis with secondary trichophytid lesions on the fingers should be considered, although the history does not suggest that likelihood. Assuming the condition to be a contact dermatitis, protective dressings, cold cream applications, and instructions regarding precautionary measures at work should suffice for clearing up this condition. If, however, the dermatitis increases in severity in spite of precautionary measures and bland protective ointment, the work should be changed. The patient should be seen promptly by a dermatologist if the measures suggested are not effective in correcting the dermatitis.

ALCOHOLISM

To the Editor:—*Please give information on hospitals or institutions where formal training in the treatment of alcoholism can be obtained.*

Thomas E. Fulghum, M.D., Augusta, Ga.

Answer.—This consultant does not know of any hospitals or institutions where training in treatment of alcoholism can be secured. Requests for accepting physicians, rehabilitation counsellors, psychiatric social workers, and psychologists on a training basis in state facilities are frequently received, but, since these facilities are not organized as training centers but rather as service centers for alcoholics and their families requesting assistance with drinking problems, they are unable to provide such

training. At a recent meeting in Washington with representatives from the National Institutes of Health and from the National Institute of Mental Health, this question was discussed and a need for training facilities for professional people interested in the problem was recognized. The present system is to select new employees with as much care as possible and then to give them on-the-job training.

CEREBRAL EMBOLISM

To the Editor:—*A 45-year-old man collapsed, with a loss of consciousness. Two days previously he had collapsed, without loss of consciousness, and had experienced a benumbed feeling of weakness on his entire left side. He had had a head cold for which nose drops containing ephedrine were used rather frequently. Forty-eight hours after using the drops the patient suddenly collapsed while at work and was admitted to a hospital, in a comatose state, with working diagnosis of auricular fibrillation and cerebral embolism. The diagnosis was based on an abnormal electrocardiogram tracing. X-ray showed a cardiac-thoracic ratio of 14 cm. to 30 cm.; right ventricular hypertrophy is minimal. There is indentation of the left ventricle (enlarged left auricle) by the esophagus. The impression given is of mitral stenosis. Did the nasal medication, more particularly the ephedrine, possibly play any part in causing the cerebral embolism?*

A. V. Spaeth, Cleveland, Ohio.

Answer.—Much study and attention have been given to the problem of causation or precipitation of embolism to the brain. In general, the specific embolic episodes seem unrelated to exogenous factors. Under the circumstances described, with the lapse of 48 hours from the use of ephedrine to the cerebral ictus, the relationship with the ephedrine certainly could not be established. In all probability, therefore, the cerebral embolus cannot be related to the use of the ephedrine.

TEACHING OF HYPNOSIS

To the Editor:—*Please give information on an ethical manner for teaching of hypnosis.*

F. P. McNalley, M.D., Clayton, Mo.

Answer.—Any course dealing with hypnotic techniques should not primarily stress the induction of hypnosis, since this is a relatively simple manner which requires little technical skill and little training. Some work on what can best be termed the motivational bases of human behavior seems to be a prerequisite. Any training should include not only techniques of induction but also indications and limitations for its use within the specific area involved. Precautions and potential dangers should be stressed. All such training should be under responsible medical (or dental) direction. Any use of hypnotic techniques should be for purposes within the area of one's specific specialty or usual medical practice and should not be beyond the range of his ordinary competence.

BROWN SUGAR

To the Editor:—*Is the ordinary brown sugar bought in the stores perfectly safe to be eaten on dry and cooked cereal, that is, eaten as bought?*

Philip G. Kitchen, M.D., Pocono Lake, Pa.

Answer.—Brown sugar is simply a partially refined white sugar. The sucrose content of brown sugar used in commerce ranges from about 85 to 90% and the mineral content from about 0.5 to 2%—the darker the color the less sucrose and the higher the mineral content. These minerals consist chiefly of calcium, phosphorus, and iron. This consultant is unaware of any evidence indicating that brown sugar, as purchased in the store, is not safe to eat. It has a characteristic taste which some people prefer and contributes small amounts of some of the minerals, as indicated above. Brown sugar may be used as is ordinary white sugar.

VITAMIN B_{12}

To the Editor:—*Has vitamin B_{12} in gelatin any real advantage over the usual preparation given by hypodermic injection in treatment of pernicious anemia?* *M.D., North Dakota.*

Answer.—There is no established advantage of vitamin B_{12} in gelatin over the conventional vitamin B_{12} preparations given parenterally. Experience and experimental studies have shown that vitamin B_{12} is stored in the body to the extent that an injection every three or four weeks provides adequate maintenance therapy in pernicious anemia.

PROFESSIONAL LIABILITY

To the Editor:—*A surgeon and an anesthesiologist recognize a situation which might possibly give rise to the filing of a claim for professional liability. The patient has consulted an attorney, but nothing more specific than that has yet occurred. Is there anything the surgeon and anesthesiologist should do at this stage?* *M.D., California.*

Answer.—There are two things which should be done immediately by each of them. First, they should give a full and complete report of the entire occurrence to the company, or companies, which are carrying their professional liability insurance. Second, they should make a full and complete report of the situation to the proper individual in their county society so that the matter can be referred to the Malpractice Review Committee, or whatever similar committee exists in their locality.

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JOURNALS ABSTRACTED IN THE CURRENT MEDICAL LITERATURE DEPARTMENT, JANUARY-APRIL 1958

Abstracts have been made of important articles in the following journals in the Current Medical Literature Department of THE JOURNAL during the past four months. Any of the journals, except those starred, will be lent by the Library of the American Medical Association to individuals in Continental United States and Canada who subscribe to its scientific periodicals and to members of the American Medical Association and its student organization. No charge is made to members, but the fee for other borrowers is 15 cents in stamps for each periodical. Three journals may be borrowed at a time for a period not exceeding five days. Thus most of these journals are accessible to the general practitioner.

*A. M. A. Archives of Dermatology. Chicago.
*A. M. A. Archives of Industrial Health. Chicago.
*A. M. A. Archives of Internal Medicine. Chicago.
*A. M. A. Archives of Neurology and Psychiatry. Chicago.
*A. M. A. Archives of Ophthalmology. Chicago.
*A. M. A. Archives of Otolaryngology. Chicago.
*A. M. A. Archives of Pathology. Chicago.
*A. M. A. Archives of Surgery. Chicago.
*A. M. A. Journal of Diseases of Children. Chicago.
Acta chirurgica scandinavica. Stockholm.
Acta medica scandinavica. Stockholm.
Acta obstetrica et gynecologica scandinavica. Stockholm.
Acta paediatrica. Uppsala.
Acta tuberculosea belgica. Brussels.
American Heart Journal. St. Louis.
American Journal of Clinical Pathology. Baltimore.
American Journal of Digestive Diseases. New York.
American Journal of Gastroenterology. New York.
American Journal of Hygiene. Baltimore.
American Journal of the Medical Sciences. Philadelphia.
American Journal of Medicine. New York.
American Journal of Obstetrics and Gynecology. St. Louis.
American Journal of Ophthalmology. Chicago.
American Journal of Pathology. Ann Arbor.
American Journal of Psychiatry. New York.
American Journal of Public Health. Albany.
American Journal of Roentgenol. and Radium Therapy. Springfield, Ill.
American Journal of Surgery. New York.
American Journal of Tropical Medicine and Hygiene. Baltimore.
American Practitioner and Digest of Treatment. Philadelphia.
American Review of Tuberculosis. New York.
American Surgeon. Baltimore.
Anales de cirugia Rosario. Argentina.
Angiology. Baltimore.
Annals of Allergy. St. Paul.
Annals of Internal Medicine. Lancaster, Pa.
Annals of Otology, Rhinology and Laryngology. St. Louis.
Annals of Rheumatic Diseases. London.
Annals of Surgery. Philadelphia.
Antibiotic Medicine. New York.
Archivos del Instituto de cardiologia de México. México, D. F.
Archivos médicos panamenos. Panama.
Archiv für klinische Chirurgie. Berlin.
Archives of Disease in Childhood. London.
Archivum chirurgicum neerlandicum. Arnhem.
Arkhiv patologii. Moscow.
Arquivos mineiros de leprologia. Belo Horizonte.
Arquivo de patologia. Lisbon.
Australian & New Zealand Journal of Surgery. Melbourne.
Australasian Annals of Medicine. Sydney.
Belgische tijdschrift voor geneeskunde. Leuven.
Blood. New York.
Brain. London.
British Heart Journal. London.
British Journal of Cancer. London.
British Journal of Ophthalmology. London.
British Journal of Radiology. London.
British Journal of Surgery. Bristol.
British Journal of Tuberculosis and Diseases of the Chest. London.
British Journal of Urology. Edinburgh.
British Journal of Venereal Diseases. London.
Bulletin of the Johns Hopkins Hospital. Baltimore.
Bulletin of the Los Angeles Neurological Society. Los Angeles.
California Medicine. San Francisco.
Canadian Anaesthetists Society Journal. Toronto.
Canadian Journal of Public Health. Toronto.
Canadian Journal of Surgery. Toronto.
Canadian Medical Association Journal. Montreal.
Cancer. Philadelphia.
Cancer Research. Chicago.
Chinese Medical Journal. Peking.
Chirurgia toracica. Rome.
Chirurg. Berlin.
Circulation. New York.
Clinica. Bologna.
Clinica pediatrica. Bologna.
Clinical Science. London.
Danish Medical Bulletin. Copenhagen.
Deutsche medizinische Wochenschrift. Stuttgart.
Diabetes. New York.
Die Medizinische. Stuttgart.
Diseases of the Chest. Chicago.
East African Medical Journal. Nairobi.
Finska läkaresällskapets handlingar. Helsinki.
Food Research. Champaign, Ill.
Fortschritte auf dem Gebiete der Röntgenstrahlen und der Nuklearmedizin. Stuttgart.
Gaceta médica de México. Mexico, D. F.
Gastroenterology. Baltimore.
Gazzetta medica italiana. Turin.
Giornale di clinica medica. Bologna.
Helvetica chirurgica acta. Basel.
Industrial Medicine and Surgery. Chicago.
International Journal of Anesthesia. Baltimore.
Journal of Allergy. St. Louis.
Journal of the American Geriatrics Society. Baltimore.
Journal of the American Medical Women's Association. New York.
Journal of the Arkansas Medical Society. Fort Smith.
Journal of Aviation Medicine. St. Paul.
Journal of Bone and Joint Surgery. Boston and London.
Journal of Chronic Diseases. St. Louis.
Journal of Clinical Endocrinology and Metabolism. Springfield, Ill.
Journal of Clinical Investigation. New York.
Journal of Experimental Medicine. New York.
Journal of the Florida Medical Association. Jacksonville.
Journal of the Indiana State Medical Association. Indianapolis.
Journal of International College of Surgeons. Chicago.
Journal of Iowa State Medical Society. Des Moines.
Journal of Kansas Medical Society. Topeka.
Journal of the Kentucky State Medical Association. Louisville.
Journal of Laboratory and Clinical Medicine. St. Louis.
Journal of Laryngology and Otology. London.
Journal of the Louisiana State Medical Society. New Orleans.
Journal of the Maine Medical Association. Brunswick.
Journal of the Medical Association of Georgia. Atlanta.
Journal of Michigan State Medical Society. St. Paul.
Journal of Neurology, Neurosurgery and Psychiatry. London.
Journal of Neurosurgery. Springfield, Ill.
Journal of Obstetrics and Gynaecology of the British Empire. London.
Journal of Pathology and Bacteriology. Edinburgh.
Journal of Pediatrics. St. Louis.
Journal of Thoracic Surgery. St. Louis.
Journal of Urology. Baltimore.
Khirurgiya. Moscow.
Klinicheskaya meditsina. Moscow.
Klinische Wochenschrift. Berlin.
Lancet. London.
Lotta contro la tubercolosi. Rome.
Maandschrift voor kindergeneeskunde. Leyden.
Medical Annals of the District of Columbia. Washington, D. C.
Medical Journal of Australia. Sydney.
Medicine. Baltimore.
Metabolism. New York.
Military Medicine. Washington, D. C.
Minerva cardioangiologica. Turin.
Minerva chirurgica. Turin.
Minerva ginecologica. Turin.
Minerva medica. Turin.
Minerva pediatrica. Turin.
Minerva stomatologica. Turin.
Minnesota Medicine. St. Paul.
Monatsschrift für Kinderheilkunde. Berlin.
Münchener medizinische Wochenschrift. Munich.
Nebraska State Medical Journal. Lincoln.
Nederlandsch tijdschrift voor geneeskunde. Haarlem.
Neurology. Minneapolis.
New England Journal of Medicine. Boston.
New York State Journal of Medicine. New York.
North Carolina Medical Journal. Winston-Salem.
Northwest Medicine. Seattle.
Nordisk medicin. Stockholm.
Obstetrics and Gynecology. New York.
Obstetricia y ginecologia latino-americanas. Buenos Aires.
Ohio State Medical Journal. Columbus.
Pediatrics. Springfield, Ill.
Pédiatrie. Lyon.
Pennsylvania Medical Journal. Harrisburg.
Plastic & Reconstructive Surgery. Baltimore.
Policlinico (sezione chirurgica). Rome.
Postgraduate Medicine. Minneapolis.
Presse médicale. Paris.
Problems of Oncology. New York.
Proceedings of the Society for Experimental Biology and Medicine. Utica.
Proceedings of Staff Meetings of Mayo Clinic. Rochester, Minn.
Psychosomatic Medicine. New York.
Quarterly Bulletin of Northwestern University Medical School. Chicago.
Quarterly Journal of Medicine. Oxford.
Radiology. Syracuse, N. Y.
Revista de la Asociación médica argentina. Buenos Aires.
Revista clinica española. Madrid.
Revista española de pediatria. Saragossa.
Revista médica de Chile. Santiago.
Revista paulista de medicina. São Paulo.
Revue française d'études cliniques et biologiques. Paris.
Revue de la tuberculose. Paris.
Riforma medica. Naples.
Rivista di clinica pediatrica. Florence.
Rivista di patologia e clinica della tubercolosi. Bologna.
Rocky Mountain Medical Journal. Denver.

Schweizerische medizinische Wochenschrift. Basel.
Schweizerische Zeitschrift für Tuberkulose und Pneumonologie. Basel.
Scottish Medical Journal. Glasgow.
Semaine des hôpitaux de Paris. Paris.
Settimana medica. Florence.
South Dakota Journal of Medicine and Pharmacy. Sioux Falls.
Southern Medical Journal. Birmingham, Ala.
Sovetskaya meditsina. Moscow.
Strahlentherapie. Munich.
Surgery. St. Louis.
Surgery, Gynecology and Obstetrics. Chicago.
Thorax. London.
Tidsskrift for den norske laegeforening. Oslo.
Tórax. Montevideo.
Tubercle. London.
Tuberkulosearzt. Stuttgart.
Ugeskrift for laeger. Copenhagen.
Vestnik khirurgii. Moscow-Leningrad.
Wiener Zeitschrift für innere Medizin und ihre Grenzgebiete. Vienna.
West Virginia Medical Journal. Charleston.
Wisconsin Medical Journal. Madison.
Zeitschrift für die gesamte innere Medizin und ihre Grenzgebiete. Leipzig.
Zeitschrift für Kinderheilkunde. Berlin.

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* Cannot be lent.

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SUBJECT INDEX

The letters used to explain in which department the matter indexed appears are as follows: "E," Editorial; "C," Correspondence; "ab," abstracts; the asterisk (*) indicates an original article in THE JOURNAL.

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Am.—American *Inst.—Institute*
A.—Association *Internat.—International*
Coll.—College *M.—Medical*
Commn.—Commission *Med.—Medicine*
Comm.—Committee *Nat.—National*
Conf.—Conference *Pharm.—Pharmaceutical*
Cong.—Congress *Phys.—Physicians*
Dist.—District *Soc.—Society*
Div.—Division *Surg.—Surgery*
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